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DIAGNOSIS OF THE CAUSES OF HEART FAILURE

LOUIS HAMMAN, M.D.*

BALTIMORE

THE aim of medical practice is the prevention and cure of disease. The function of diagnosis is to direct and guide treatment. Unfortunately the refinements of diagnosis have far outrun the inventions of treatment, and we find ourselves in the predicament of being able to recognize and classify many morbid conditions the natural course of which we can modify little if at all. This state of affairs has led to criticism of our preoccupation with diagnosis which has no immediate application in treatment. We are told that diagnosis has become an academic diversion, a form of intellectual coxcombry, an exhibition of professional vanity.

I confess that I smart under these reproaches, and therefore I suspect there must be some truth in them, even though I am not at a loss to suggest many cogent reasons to prove them undeserved and misleading. Nevertheless, I have the uncomfortable feeling that I should offer an apology for venturing to discuss the diagnosis of the causes of heart failure. In the first place, the correctness of the diagnosis can be established only at the postmortem examination, when the patient is beyond the reach of help through our diagnostic finesse. In the second place, we cannot even urge that the diagnostic skill acquired by the demonstration of our successive mistakes fits us in a striking degree to benefit patients who may subsequently come under our care. I do not mean to imply that distinctions are without any practical significance. For instance, to recognize early that heart disease is syphilitic or due to pericardial adhesions is of some consequence to the patient. But when serious failure has once set in, treatment is the same whatever may be the cause of the failure. I shall offer no defense against these views. Certainly it can do no harm to sharpen our wits through exercise, and the practice may train us to perform better on some critical occasion. At any rate it will instruct us in the natural course of disease. I have no objection, however, to your looking upon this presentation as a form of idle amusement. Doctors need recreation; and diagnosis, even when not immediately fruitful in practice, is at least a fascinating diversion.

In diagnosis our ultimate aim is to reach an etiologic classification. On the other hand, such a diagnosis is often incomplete, and must usually be amplified by an anatomic and a functional one. Merely to say that a patient has rheumatic heart disease, for example, leaves us in a questioning frame of mind. Rheumatic fever affects the heart in many different ways; the lesions may accompany an active infection or be the results of infection long since overcome; the defect noted in the heart may produce no symptoms whatsoever or may cause the grave symptoms of failure. Moreover, we can never make an etiologic diagnosis directly. First we consider the functional disturbances which indicate the presence of heart disease; then from an analysis of the symptoms and the results of the examination we determine the location and extent of the anatomic lesion that may be present; finally from these conclusions and all other related facts we decide upon the etiologic diagnosis. There are many occasions when after a brief examination of the heart the physician may proclaim at once the correct etiologic diagnosis, but when he does so it must be borne in mind that experience has trained him to jump to the conclusion over a number of logical steps which he apparently ignores. For most of us it is safer to heed the steps and climb them cautiously.

As we observe patients with the symptoms of heart disease on hospital wards, in outpatient departments and in private practice we notice at once that most of them have valvular lesions or hypertension. These are the usual precursors of heart failure. Less frequently we discover evidence of pericardial effusion or adhesions, of congenital defects and of hypertension in the pulmonary circulation. In a further number of cases the symptoms force us to conclude that the heart has failed as

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the result of destructive disease of the heart muscle itself. All but a few cases of heart failure fit into one or more of these six categories: valvular heart disease; hypertension; pericardial effusion or adhesions; congenital anomalies; pulmonary hypertension; myocardial disease.

The first step in diagnosis is to decide in which of these groups the case under consideration belongs. The heart begins to fail when the burden of work imposed upon it is greater than its capacity. Usually the increased burden is due to a mechanical obstacle in the circulation: defective action of the valves, increased resistance to the blood flow, or a hindrance to the filling or emptying of the heart. Less often it is due to changes in the heart muscle which destroy a large number of the fibers or render them functionally incompetent, under which circumstances the heart fails even though the demands upon it are only the minimal ones required to maintain a normal circulation. For purposes of diagnosis it is expedient and convenient to contrast heart failure caused by mechanical impediment to the circulation with that caused by disease of the myocardium. We must nevertheless bear in mind that in both events the heart fails for the same reason. A heart composed of healthy muscle fibers, in so far as we may speak of them as healthy in the light of our present methods of examination, will fail when an increased burden of work is put upon it over a long period of time; and a heart with damaged muscle will fail for the same reason, even though the circulatory demands be unaltered, because minimal circulatory demands may exceed the power of the remaining healthy muscle fibers. In either condition functionally active fibers respond to the increased work required of them by enlarging, and the opinion is becoming more and more prevalent that hypertrophied fibers are no longer healthy — or let us say are in a position of great vulnerability — so far as their functional capacity is concerned. According to this view the muscle fiber hypertrophies because more work is demanded of it; having done so, it is capable of more work and the response is therefore, at least temporarily, a beneficial one; nevertheless, with this change is implanted the seed of future disintegration. Investigations now being pursued will in the end, I have no doubt, yield a satisfactory explanation of the heretofore mysterious fact that hypertrophied hearts which for years may have satisfactorily sustained the circulation against a heavy burden of increased work do finally become exhausted even though with our present methods of examination the muscle fibers appear to be structurally sound.

Let me emphasize that the classification I have proposed as a convenient scheme to guide our analysis of the causes of heart failure is in many ways an artificial one. Disease does not operate in a fashion so cut and dried as this. As you well know, situations are often complicated and frequently two, sometimes three, of the conditions here considered apart unite to bring about heart failure. Even under these complicated circumstances, however, close attention to the clinical facts will often enable us to diagnose correctly the combinations which have occurred.

VALVULAR HEART DISEASE

It is often extraordinarily difficult to decide whether there is structural disease of the valves; yet the decision is of fundamental importance in diagnosis. There are certain murmurs which allow us to say confidently that the valves are incompetent, but we may be unable to tell whether the incompetency is due to disease of the valves or to conditions causing what are called functional murmurs. If so, our decision must be reached by a careful consideration of alterations which may have occurred in the circulation: changes in the quality of the pulse and of the size of the heart; if the latter is enlarged, its configuration and the relative preponderance of the right or left ventricle, associated valvular and other lesions, the relative tension in the peripheral and pulmonary circulations as revealed by the intensity of the second sounds, and lastly, the nature of some associated disease. The last-named consideration is always important and sometimes decisive; for instance, in children every febrile disturbance is generally accompanied by a mitral systolic murmur, but if the febrile disease is rheumatic fever the chances are greatly in favor of infection of the mitral valve.

Uncertainty about the significance of a murmur is especially great when it is a mitral systolic one. This is a matter of daily experience, which has taught us never to interpret this murmur as evidence of organic valvular disease unless the associated circumstances force the conclusion upon us. Nevertheless the decision is an important one, for mitral insufficiency caused by structural disease of the valves is nearly always due to rheumatic fever or bacterial endocarditis. Very often, autopsy discloses plaques of sclerosis on the mitral valves of persons over fifty, but only rarely do these lesions produce deformity of the valves.

While the signs of mitral stenosis, when clearly developed, are distinctive, it may be difficult to decide whether the stenosis is relative or is due to an organic lesion. The difficulty is particularly great in the presence of aortic insufficiency. Very frequently we are called upon to distinguish be-

tween syphilitic and rheumatic aortic insufficiency. If we can say confidently that there is an associated organic stenosis of the mitral valve, the lesion is undoubtedly rheumatic. But only rarely can we confidently come to this conclusion, and the prediction of experienced physicians is often wrong.

An aortic systolic murmur nearly always occurs with aortic insufficiency. An isolated aortic systolic murmur is heard frequently in patients of over fifty. This murmur is often regarded as a functional one due to dilatation of the aorta. Sometimes this mechanism must be invoked to explain an aortic systolic murmur, but as my experience grows I find it necessary less and less frequently to do so, because usually some slight deformity of the valves is found at autopsy. When patients are followed over many years, a soft aortic systolic murmur heard over the heart of an adult may gradually become louder and rougher, a thrill may appear, characteristic changes may occur in the pulse, and finally the patient may die of congestive heart failure, or may die suddenly before pronounced symptoms of failure set in. The condition is frequently overlooked, especially at an early stage. The postmortem examination reveals marked distortion of the valves from thickening and calcification. The etiology is obscure. Most Boston physicians and pathologists regard the lesions as rheumatic; elsewhere they are looked upon as degenerative, and of the nature of arteriosclerosis; we at Baltimore are convinced that some of them are cases of healed bacterial endocarditis.

An aortic diastolic murmur, with rare exceptions, indicates structural disease of the aortic valves. Cases of aortic insufficiency may be grouped as due to one of the following conditions: rheumatic fever, syphilis, bacterial endocarditis, arteriosclerosis, rupture of an aortic cusp, congenital malformations and relative insufficiency of the valves.

The first three are common causes, the last four exceptional ones.

The distinction between rheumatic and syphilitic aortic insufficiency is usually easy, but at times may be very difficult. The chief distinguishing points are these:

1. Symptoms in rheumatic cases generally begin before twenty years of age and nearly always before thirty. In syphilitic cases symptoms seldom begin before thirty.

2. A history of one or more definite attacks of rheumatic fever in childhood is important evidence in favor of the aortic insufficiency's being rheumatic.

3. A positive Wassermann test is equally strong evidence in favor of syphilis. Nevertheless, about 15 per cent of the cases of syphilitic aortic insufficiency show a negative reaction, and occasionally a patient with rheumatic aortic insufficiency shows a positive one.

4. Patients with rheumatic aortic insufficiency usually live many years after the lesion has been discovered; even after a period of myocardial insufficiency has occurred they sometimes recover under treatment and years pass before another attack of failure comes on. In contrast to this, when symptoms of heart failure once appear in patients with syphilitic aortic insufficiency their recovery is usually incomplete and of short duration. They seldom live longer than five years after the aortic insufficiency has developed.

5. The association of organic mitral stenosis with aortic insufficiency makes the diagnosis of a rheumatic lesion certain. But it must again be pointed out how extremely difficult it is in the presence of aortic insufficiency to be confident that an organic mitral stenosis exists.

Bacterial endocarditis frequently occurs in association with rheumatic aortic insufficiency, but very rarely in association with syphilitic aortitis. I have been much impressed by the many cases of primary bacterial endocarditis affecting the aortic valves which are discovered at autopsy although they may not have been suspected during life.

In elderly people it is not uncommon to hear a faint diastolic murmur to the left of the sternum, often intermittently, in the absence of any of the circulatory manifestations of aortic insufficiency. At postmortem examination slight deformity of the valves is found to be due to arteriosclerosis.

Rupture of a normal valve from trauma or violent exertion occurs occasionally. Nearly always the ruptured or torn valve is diseased. Usually the disease is bacterial endocarditis.

Congenital malformations seldom produce an aortic diastolic murmur. I recall seeing only one case, and even in that there was some question about the origin of the murmur. There were four aortic valves, so situated as to cause a slight aortic stenosis and probably also a slight aortic insufficiency. Pulmonary diastolic murmurs are commoner with congenital defects, and these may be erroneously regarded as aortic in origin.

Relative aortic insufficiency may occur with pronounced hypertension. Under these circumstances the murmur is faint and there are none of the circulatory manifestations of aortic insufficiency. A loud diastolic murmur accompanied by violent throbbing of the peripheral arteries and an extremely collapsible pulse sometimes occur with a dissecting aneurysm. The clinical picture is characteristic, and the diagnosis can easily be made by one familiar with it.

HYPERTENSIVE HEART DISEASE

Hypertension is a frequent cause of heart failure. The association is usually evident and presents no difficulty in diagnosis. Nevertheless, when a pa-

tient is first seen after the symptoms of failure have become pronounced, errors in interpretation are often made. It must be remembered that with heart failure the blood pressure nearly always rises, to return to a lower or a normal level when circulatory efficiency is re-established.

PERICARDIAL DISEASE

It is particularly important to recognize pericardial effusion or adhesions which cause heart failure, because under these circumstances the condition may be cured. Most cases of rapid and decided changes in the size of the heart are due to pericardial effusion. Pericardial adhesions, particularly the constricting form, may present great difficulty in diagnosis and are often overlooked. I do not wish to discuss the problem of diagnosis but merely to point out that the problem exists and often goes unsolved.

CONGENITAL LESIONS

Congenital lesions may generally be recognized by the unusual quality and position of the murmurs. It may be extremely difficult to predict the exact nature of the malformation, even though a malformation may be apparent. Now and again, however, the attempted differentiation of an acquired lesion and a congenital lesion gives rise to complete difference of opinion. In my experience more errors are made in calling acquired lesions congenital than vice versa.

PULMONARY HYPERTENSION

Hypertension in the pulmonary circulation may conveniently be divided into primary and secondary forms. The division is important for diagnosis, since only the primary form is an independent condition. Primary pulmonary hypertension is due to obstruction in the arterial blood flow and may follow sclerotic changes in the pulmonary artery, emphysema, pulmonary fibrosis or thrombosis of the pulmonary artery. Secondary pulmonary hypertension is due to congestion in the pulmonary veins and usually follows obstruction to the venous flow of blood through the heart, for example, in mitral stenosis and in failure of the left ventricle. The diagnosis of pulmonary hypertension as a cause of heart failure is made only in the primary form; in the secondary form the diagnosis is made of the condition to which it is secondary.

MYOCARDIAL DISEASE

If the evidence justifies us in excluding these five divisions as the cause of heart failure, we may assume that the cause is disease of the myocardium. The myocardial disease may be due to intoxication, to infection, to degeneration secondary to partial or complete occlusion of the coronary arteries, to dietary deficiency or to infiltration of the heart by a tumor. The most conspicuous illustration of intoxication occurs in diphtheria. On the other hand, in this disease patients usually recover completely from the myocardial damage; a small number die suddenly; congestive failure almost never occurs. The infectious diseases vary widely in the frequency with which they attack the heart muscle. For instance, rheumatic fever, typhus fever and scarlet fever affect it frequently; syphilis rather rarely; pneumonia, typhoid fever, tuberculosis, almost never to a degree which seriously impairs its function. Not infrequently there is extensive myocardial infiltration for which no satisfactory cause can be discovered. Myocardial degeneration due to partial or complete ischemia usually follows coronary arteriosclerosis; occasionally it is associated with coronary embolism or occlusion of the coronary arteries at their mouths. Recently, dietary deficiency has been invoked as an explanation of heart failure occurring under puzzling circumstances when no other altogether satisfactory cause can be found. These cases are described as beriberi heart and are sometimes successfully treated by the injection of vitamin B. Tumors often invade the heart by metastasis or by direct extension. Pulmonary carcinoma especially often attacks the pericardium and may grow into the heart. But the symptoms of heart failure are seldom produced as the result of the invasion.

Bearing in mind these brief preliminary remarks, we may now apply them to the diagnosis of actual cases of heart failure. The situations selected were not unusually difficult, although they presented definite problems in diagnosis.

CASE REPORTS

Case 1 (Unit No. 104747). A 54-year-old Negress entered the hospital September 13, 1936, complaining of shortness of breath and swelling of the abdomen and legs. For a year or more she had noticed difficulty in breathing on exertion, which had gradually become more pronounced. Two months before admission her legs had begun to swell and a little later the abdomen had become prominent. From then on the shortness of breath and the swelling had gradually increased.

Physical examination showed an obese woman, propped up in bed, with moderate dyspnea. There was a little cyanosis. The legs, thighs, sacral region and abdominal wall were swollen with edema. There was fluid in the left pleural cavity and abundant ascites. Over the lower lobe of the right lung there were numerous moist rales. The liver extended 4 cm. below the costal margin. The heart was moderately enlarged, particularly to the left. A soft systolic murmur was heard at the apex and over the body of the heart. The second aortic sound was accentuated and ringing in quality. The regular rhythm was disturbed by the occurrence of many extrasystoles. The peripheral vessels were only a little thickened.

The Wassermann reaction on the blood serum was negative. The urine on admission contained a trace of albumin, later none. A phenolsulfonephthalein test of renal function gave an excretion of 75 per cent in 2 hours. An electrocardiogram revealed no gross abnormality.

The patient responded satisfactorily to treatment. The edema soon disappeared, and she was comfortable when she left the hospital October 25. The blood pressure, which on admission was 180/120, fell to 137/76 as the patient improved. When she was better and up and about the ward, it rose to 182/98.

The patient was admitted to the hospital a second time on April 13, 1937. During the intervening 6 months she had at first been reasonably well, although always short of breath on exertion. The shortness of breath had gradually become more pronounced, and later, edema had returned. The blood pressure, taken on one occasion in the outpatient clinic, was 194/130.

On this admission the patient was more decidedly short of breath than she had been at the first admission, and the usual signs of chronic passive congestion were well developed. The breathing was Cheyne-Stokes in character. The heart was as it had been before except that periods of auricular fibrillation occurred from time to time.

The patient failed to improve satisfactorily under treatment. She was better for a while, but the symptoms returned and she became apathetic and somnolent. On May 3, when she was brighter than she had been and seemed not so ill, she died suddenly. The blood pressure on admission was 185/115. Later it fell, and on one occasion it was as low as 125/80.

The urine contained a large amount of albumin on admission, later only a trace.

Comment. A cursory review of these clinical records might have led anyone to make a diagnosis of hypertensive cardiovascular disease. On a number of occasions the blood pressure was considerably elevated, there was cardiac enlargement, predominantly of the left ventricle, and the patient died of heart failure about two years after the onset of the symptoms of myocardial insufficiency. All this is quite typical of what commonly occurs with hypertension. However, a more careful consideration of the evidence would throw doubt upon this hasty conclusion. Although the blood pressure was elevated on both occasions when the patient entered the hospital, it fell to a normal level when the symptoms of failure improved. This is not the way an established hypertension behaves, whereas this is precisely what often happens when hypertension is a temporary accompaniment of heart failure. More than this, the patient did not have the degree of arterial change usually associated with hypertension of long duration. It therefore seems wise to consider at least the possibility that there may have been some other cause for the heart failure. Under the circumstances, the only other conceivable possibility was extensive damage to the heart muscle occasioned by sclerosis of the coronary arteries. In a word, the choice consisted in deciding between hypertensive heart disease and arteriosclerotic heart disease. The diagnosis made on the ward was hypertensive heart disease, but I was so impressed by the behavior of the blood pressure that I predicted that the postmortem would reveal the characteristic changes of coronary arteriosclerosis. At autopsy the heart was moderately hypertrophied and enlarged, weighing 460 gm. Both the right and the left ventricular walls were hypertrophied. Throughout the walls of the left ventricle many small white scars were visible. At the apex of the left ventricle a firm thrombus was adherent to the wall. The valves displayed no significant change. The coronary arteries showed pronounced arteriosclerotic changes in the smaller branches, with narrowing of the lumen but nowhere complete occlusion.

In sections examined by microscope were seen numerous scars scattered throughout the myocardium, typical of those produced by coronary arteriosclerosis. The arterioles observed in the various organs showed no thickening of their walls, which plainly indicated that there had been no long-continued hypertension during life.

Case 2 (Unit No. 130975). A 39-year-old white man entered the hospital in 1924 complaining of rheumatism. When 22 years of age he had contracted a chancre, followed by a syphilitic cutaneous eruption. He had been insufficiently treated. At 35 he had had syphilitic iritis. A Wassermann test on admission was positive; the spinal fluid was normal. Seven years later he was treated by a private physician and received twenty-four intramuscular injections. At the end of this course of treatment the Wassermann reaction was still positive. He was admitted to the hospital again in April, 1934. At that time he stated that during the preceding 6 or 7 months he had had frequent attacks of severe substernal pain, lasting for 3 or 4 minutes. The detailed account of the symptoms left no doubt that these were attacks of angina pectoris. During the 3 or 4 months before admission he had become increasingly short of breath. From the time of this admission he was almost constantly under observation on the wards of the hospital and in the outpatient department.

The essential features of his condition were as follows. All the characteristic manifestations of a high grade of aortic insufficiency were present. The symptoms of myocardial insufficiency, once begun, varied in intensity, but never completely disappeared during the 4 years he was under observation. A conspicuous feature of the illness was the frequent recurrence of severe attacks of substernal pain, in all details characteristic of angina pectoris. These attacks were relieved by nitroglycerin. Numerous electrocardiograms showed pronounced changes. There was always prolongation of the auriculoventricular conduction, and at times complete heart block. Some of the records showed changes indicative of bundle-branch block. The QRS interval was sometimes prolonged and there was slurring of these complexes. There was a prominent Q wave in Lead 3. The patient was very sensitive to digitalis so that full doses of the drug could not be given.

On the evening of February 6, 1938, the patient had an attack of excruciating pain under the sternum, radiating down the left arm. The pain continued unabated, and at 1 a. m. he was brought to the hospital. Upon arrival he was desperately ill with the usual symptoms of shock. The pulse was very rapid; the systolic blood pressure,

tient is first seen after the symptoms of failure have become pronounced, errors in interpretation are often made. It must be remembered that with heart failure the blood pressure nearly always rises, to return to a lower or a normal level when circulatory efficiency is re-established.

PERICARDIAL DISEASE

It is particularly important to recognize pericardial effusion or adhesions which cause heart failure, because under these circumstances the condition may be cured. Most cases of rapid and decided changes in the size of the heart are due to pericardial effusion. Pericardial adhesions, particularly the constricting form, may present great difficulty in diagnosis and are often overlooked. I do not wish to discuss the problem of diagnosis but merely to point out that the problem exists and often goes unsolved.

CONGENITAL LESIONS

Congenital lesions may generally be recognized by the unusual quality and position of the murmurs. It may be extremely difficult to predict the exact nature of the malformation, even though a malformation may be apparent. Now and again, however, the attempted differentiation of an acquired lesion and a congenital lesion gives rise to complete difference of opinion. In my experience more errors are made in calling acquired lesions congenital than vice versa.

PULMONARY HYPERTENSION

Hypertension in the pulmonary circulation may conveniently be divided into primary and secondary forms. The division is important for diagnosis, since only the primary form is an independent condition. Primary pulmonary hypertension is due to obstruction in the arterial blood flow and may follow sclerotic changes in the pulmonary artery, emphysema, pulmonary fibrosis or thrombosis of the pulmonary artery. Secondary pulmonary hypertension is due to congestion in the pulmonary veins and usually follows obstruction to the venous flow of blood through the heart, for example, in mitral stenosis and in failure of the left ventricle. The diagnosis of pulmonary hypertension as a cause of heart failure is made only in the primary form; in the secondary form the diagnosis is made of the condition to which it is secondary.

MYOCARDIAL DISEASE

If the evidence justifies us in excluding these five divisions as the cause of heart failure, we may assume that the cause is disease of the myocardium. The myocardial disease may be due to intoxication, to infection, to degeneration secondary to partial or complete occlusion of the coronary arteries, to dietary deficiency or to infiltration of the heart by a tumor. The most conspicuous illustration of intoxication occurs in diphtheria. On the other hand, in this disease patients usually recover completely from the myocardial damage; a small number die suddenly; congestive failure almost never occurs. The infectious diseases vary widely in the frequency with which they attack the heart muscle. For instance, rheumatic fever, typhus fever and scarlet fever affect it frequently; syphilis rather rarely; pneumonia, typhoid fever, tuberculosis, almost never to a degree which seriously impairs its function. Not infrequently there is extensive myocardial infiltration for which no satisfactory cause can be discovered. Myocardial degeneration due to partial or complete ischemia usually follows coronary arteriosclerosis; occasionally it is associated with coronary embolism or occlusion of the coronary arteries at their mouths. Recently, dietary deficiency has been invoked as an explanation of heart failure occurring under puzzling circumstances when no other altogether satisfactory cause can be found. These cases are described as beriberi heart and are sometimes successfully treated by the injection of vitamin B. Tumors often invade the heart by metastasis or by direct extension. Pulmonary carcinoma especially often attacks the pericardium and may grow into the heart. But the symptoms of heart failure are seldom produced as the result of the invasion.

Bearing in mind these brief preliminary remarks, we may now apply them to the diagnosis of actual cases of heart failure. The situations selected were not unusually difficult, although they presented definite problems in diagnosis.

CASE REPORTS

Case 1 (Unit No. 104747). A 54-year-old Negress entered the hospital September 13, 1936, complaining of shortness of breath and swelling of the abdomen and legs. For a year or more she had noticed difficulty in breathing on exertion, which had gradually become more pronounced. Two months before admission her legs had begun to swell and a little later the abdomen had become prominent. From then on the shortness of breath and the swelling had gradually increased.

Physical examination showed an obese woman, propped up in bed, with moderate dyspnea. There was a little cyanosis. The legs, thighs, sacral region and abdominal wall were swollen with edema. There was fluid in the left pleural cavity and abundant ascites. Over the lower lobe of the right lung there were numerous moist rales. The liver extended 4 cm. below the costal margin. The heart was moderately enlarged, particularly to the left. A soft systolic murmur was heard at the apex and over the body of the heart. The second aortic sound was accentuated and ringing in quality. The regular rhythm was disturbed by the occurrence of many extrasystoles. The peripheral vessels were only a little thickened.

The Wassermann reaction on the blood serum was negative. The urine on admission contained a trace of albumin, later none. A phenolsulfonephthalein test of renal function gave an excretion of 75 per cent in 2 hours. An electrocardiogram revealed no gross abnormality.

The patient responded satisfactorily to treatment. The edema soon disappeared, and she was comfortable when she left the hospital October 25. The blood pressure, which on admission was 180/120, fell to 137/76 as the patient improved. When she was better and up and about the ward, it rose to 182/98.

The patient was admitted to the hospital a second time on April 13, 1937. During the intervening 6 months she had at first been reasonably well, although always short of breath on exertion. The shortness of breath had gradually become more pronounced, and later, edema had returned. The blood pressure, taken on one occasion in the outpatient clinic, was 194/130.

On this admission the patient was more decidedly short of breath than she had been at the first admission, and the usual signs of chronic passive congestion were well developed. The breathing was Cheyne-Stokes in character. The heart was as it had been before except that periods of auricular fibrillation occurred from time to time.

The patient failed to improve satisfactorily under treatment. She was better for a while, but the symptoms returned and she became apathetic and somnolent. On May 3, when she was brighter than she had been and seemed not so ill, she died suddenly. The blood pressure on admission was 185/115. Later it fell, and on one occasion it was as low as 125/80.

The urine contained a large amount of albumin on admission, later only a trace.

Comment. A cursory review of these clinical records might have led anyone to make a diagnosis of hypertensive cardiovascular disease. On a number of occasions the blood pressure was considerably elevated, there was cardiac enlargement, predominantly of the left ventricle, and the patient died of heart failure about two years after the onset of the symptoms of myocardial insufficiency. All this is quite typical of what commonly occurs with hypertension. However, a more careful consideration of the evidence would throw doubt upon this hasty conclusion. Although the blood pressure was elevated on both occasions when the patient entered the hospital, it fell to a normal level when the symptoms of failure improved. This is not the way an established hypertension behaves, whereas this is precisely what often happens when hypertension is a temporary accompaniment of heart failure. More than this, the patient did not have the degree of arterial change usually associated with hypertension of long duration. It therefore seems wise to consider at least the possibility that there may have been some other cause for the heart failure. Under the circumstances, the only other conceivable possibility was extensive damage to the heart muscle occasioned by sclerosis of the coronary arteries. In a word, the choice consisted in deciding between hypertensive heart disease and arteriosclerotic heart disease. The diagnosis made on the ward was hypertensive heart disease, but I was so impressed by the behavior of the blood pressure that I predicted that the postmortem would reveal the characteristic changes of coronary arteriosclerosis. At autopsy the heart was moderately hypertrophied and enlarged, weighing 460 gm. Both the right and the left ventricular walls were hypertrophied. Throughout the walls of the left ventricle many small white scars were visible. At the apex of the left ventricle a firm thrombus was adherent to the wall. The valves displayed no significant change. The coronary arteries showed pronounced arteriosclerotic changes in the smaller branches, with narrowing of the lumen but nowhere complete occlusion.

In sections examined by microscope were seen numerous scars scattered throughout the myocardium, typical of those produced by coronary arteriosclerosis. The arterioles observed in the various organs showed no thickening of their walls, which plainly indicated that there had been no long-continued hypertension during life.

Case 2 (Unit No. 130975). A 39-year-old white man entered the hospital in 1924 complaining of rheumatism. When 22 years of age he had contracted a chancre, followed by a syphilitic cutaneous eruption. He had been insufficiently treated. At 35 he had had syphilitic iritis. A Wassermann test on admission was positive; the spinal fluid was normal. Seven years later he was treated by a private physician and received twenty-four intramuscular injections. At the end of this course of treatment the Wassermann reaction was still positive. He was admitted to the hospital again in April, 1934. At that time he stated that during the preceding 6 or 7 months he had had frequent attacks of severe substernal pain, lasting for 3 or 4 minutes. The detailed account of the symptoms left no doubt that these were attacks of angina pectoris. During the 3 or 4 months before admission he had become increasingly short of breath. From the time of this admission he was almost constantly under observation on the wards of the hospital and in the outpatient department.

The essential features of his condition were as follows. All the characteristic manifestations of a high grade of aortic insufficiency were present. The symptoms of myocardial insufficiency, once begun, varied in intensity, but never completely disappeared during the 4 years he was under observation. A conspicuous feature of the illness was the frequent recurrence of severe attacks of substernal pain, in all details characteristic of angina pectoris. These attacks were relieved by nitroglycerin. Numerous electrocardiograms showed pronounced changes. There was always prolongation of the auriculoventricular conduction, and at times complete heart block. Some of the records showed changes indicative of bundle-branch block. The QRS interval was sometimes prolonged and there was slurring of these complexes. There was a prominent Q wave in Lead 3. The patient was very sensitive to digitalis so that full doses of the drug could not be given.

On the evening of February 6, 1938, the patient had an attack of excruciating pain under the sternum, radiating down the left arm. The pain continued unabated, and at 1 a. m. he was brought to the hospital. Upon arrival he was desperately ill with the usual symptoms of shock. The pulse was very rapid; the systolic blood pressure,

which before had always been elevated, was 90. His condition remained but little altered for 12 hours. At 1 p. m. he suddenly grew worse. He complained of intense substernal pain, the pulse became very feeble, the cyanosis deepened, and he gradually lapsed into coma and a little later died.

Comment. From this clinical record we must explain three points: the character of the aortic insufficiency, the cause of the angina pectoris and the final symptoms of coronary occlusion, and the meaning of the electrocardiographic changes.

There was no other way to explain the aortic insufficiency than by assuming that it was due to syphilitic aortitis. The whole clinical course was quite characteristic, and this diagnosis could be made with confidence. This being so, it seemed desirable to explain all the clinical manifestations on the basis of the syphilitic infection. In an effort to do so we surmised that the syphilitic process in the aorta had gradually encroached upon the mouths of the coronary arteries, narrowing their lumens and thus causing the attacks of angina pectoris, and that finally it had completely occluded one of the coronaries, thus producing the characteristic symptoms of coronary occlusion which immediately preceded death. Since the electrocardiograms exhibited changes characteristic of severe myocardial damage, we were further obliged to conclude that extensive syphilitic disease had attacked the heart muscle, in the form either of diffuse infiltration and fibrosis or of localized scarring secondary to gumma formation. This prediction was certainly quite reasonable. Nevertheless it should be pointed out that it was not entirely consistent with the clinical symptoms. In syphilitic aortitis such persistent pain as this patient had over such a long period of time is unusual, and it is almost equally unusual for the patient to die with the typical symptoms of coronary occlusion. These manifestations and the associated electrocardiographic changes are certainly much more distinctive of arteriosclerotic heart disease, and I think no one would have hesitated to make this diagnosis had it not been for the clear evidence of the presence of syphilitic aortitis. Here, then, we were presented with the choice of deciding whether all the changes were due to syphilis, or whether the aortic insufficiency was due to syphilis but the arterial and myocardial changes to arteriosclerosis.

I chose to explain all the lesions as syphilitic manifestations. In making this choice I was greatly influenced by a case we had had a few months before. A colored man of forty-nine died with the symptoms of coronary occlusion seven weeks after the onset of typical attacks of angina pectoris. The aorta was dilated and a faint diastolic murmur was heard to the left of the sternum. A Wassermann test was strongly positive. There was no doubt that the patient had syphilitic aortitis, but I predicted that the coronary symptoms would be explained by arteriosclerotic change. At autopsy it was found that the syphilitic process had greatly narrowed the mouths of the coronary arteries, and it seemed likely that at the end a thrombus had occluded the right coronary, although no thrombus could be demonstrated. As regards the case we are now discussing, it is interesting to point out that every fourth-year student in the medical group voted in favor of arteriosclerosis.

At autopsy the heart was found to be greatly enlarged, weighing 1020 gm. Both ventricles were hypertrophied, the left much more so than the right. The aorta showed extensive changes, a mixture of syphilitic and arteriosclerotic lesions. The aortic valves were deformed in the manner characteristic of syphilis. The mouths of the coronary arteries were little if at all narrowed. In contrast, along the courses of the arteries were many plaques of arteriosclerosis. The walls of the arteries were greatly thickened, and in many the lumen was almost completely obliterated. Microscopic examination of sections of the heart muscle showed extensive scarring typical of coronary sclerosis. The case was therefore an interesting combination of syphilitic aortic insufficiency and coronary arteriosclerosis, each condition contributing its share to the clinical symptoms.

Case 3 (Unit No. 68113). A 36-year-old Negro, a laborer, was admitted to the hospital desperately ill on March 2, 1936, and died the following day. From boyhood he had always been extremely fat. In 1927 he had contracted syphilis; for 2 years thereafter had attended the syphilis clinic irregularly and had received a certain amount of treatment.

When the patient entered the hospital on March 2 he said he had been short of breath on exertion for over 2 years. About 3 months before admission the shortness of breath had become more pronounced and swelling of the legs had come on. The shortness of breath and swelling of the legs and body had steadily increased.

Physical examination showed a very obese man, tremendously swollen with edema. The heart was greatly enlarged, the sounds were clear, and there was a marked presystolic gallop. There was no thickening of the peripheral vessels; the blood pressure was 130/70. In addition to the subcutaneous edema there were ascites and left pleural effusion.

For a while the patient was somewhat more comfortable, but the following morning he became unconscious, pulmonary edema rapidly developed, and a little later he died.

Comment. It is quite clear from this brief clinical record that none of the mechanical features which usually induce heart failure were present, for there was no evidence whatsoever of a valvular lesion, of hypertension, of pericardial disease, of a congenital defect or of hypertension in

the pulmonary circulation. Without doubt the cause of the heart failure must have resided in the heart muscle, and the diagnostic problem was to decide upon the nature of the myocardial damage. Three possibilities presented themselves: coronary arteriosclerosis, inflammatory disease of the myocardium and degeneration of the myocardium.

Arteriosclerotic heart disease was altogether unlikely. The age of the patient, the absence of any evidence of arterial disease and the character of the symptoms were all strong points against it. Among the inflammatory lesions were considered only those of rheumatic, of syphilitic and of unknown etiology. The complete absence of any indication of pericardial or endocardial involvement is very uncommon in rheumatic hearts. It might be necessary to present a long array of facts to convince skeptics that severe myocardial disease may be due to syphilis. All I need say is that at Baltimore we are convinced that it does frequently occur, although nearly always in Negroes. Moreover, the symptoms may be very like those presented by this patient. Now and again we see hypertrophied hearts that have failed which show extensive myocardial infiltration, the cause of which is unknown. Finally, there are cases in which the most careful microscopical examination of the heart fails to demonstrate any concrete abnormality to explain the hypertrophy and the failure. After a detailed discussion of these possibilities it was decided that the evidence in the case just reported favored the diagnosis of syphilitic myocarditis.

At the postmortem examination the heart was found to be greatly hypertrophied and dilated. The aorta was normal. There was no deformity of the valves whatever. The coronary arteries were not diseased. There was no infiltration or scarring of the myocardium.

Cases such as these are a sore trial to the pathologist as well as to the clinician. The pathologist expressed the opinion that the above case might be one of work hypertrophy, secondary to the increased burden put upon the heart by the great obesity. As you well know, here in Boston, evidence has been accumulated which indicates that some of these cases are the result of vitamin B deficiency, a condition spoken of as beriberi heart.

Case 4 (Unit No. 53593). A 55-year-old Negress, a cook, entered the hospital February 4, 1934, complaining of pain and fullness in the chest. She had always been strong and healthy, and her illness came on very suddenly. Four weeks before admission, while working in the kitchen, she suddenly had a feeling of fullness in the chest and throat. The sensation passed off in a few minutes. From then on she noticed shortness of breath on exertion. On the day of admission, when standing before the stove, she was seized with excruciating pain under the sternum, accompanied by shortness of breath and a sense of choking. She was at once brought to the hospital.

Physical examination showed an obese woman, propped up in bed, with great respiratory distress, complaining constantly of pain and oppression under the sternum. The heart was moderately enlarged. Loud systolic and diastolic murmurs were heard over the whole heart, being loudest at the base. There was violent pulsation in the peripheral arteries, especially in the episternal notch and in the right subclavian artery, which seemed to be dilated. The pulse was regular, equal at the two wrists and collapsible. The blood pressure was 200/110. At the bases of the lungs were heard numerous moist rales. The liver was much enlarged. The urine contained some albumin. The legs were slightly swollen with edema.

The blood was normal except that the leukocyte count was slightly elevated. A Wassermann test on the blood serum was strongly positive. A roentgenogram showed great dilatation of the heart and aorta, with slight displacement of the trachea to the right. An electrocardium read as follows: levogram; slight slurring of QRS in Lead 3; T wave sharply inverted in Lead 1; PR interval 0.2 second.

On the afternoon of the day of admission the patient had an extremely severe attack of substernal pain during which she said she felt as though her chest were being torn open. Her condition did not improve. She had frequent access of great substernal pain. The murmurs over the heart became louder. On February 20, or 16 days after entering the hospital, she died suddenly.

Comment. This is an interesting case, because the symptoms were so typical that anyone familiar with them would not have hesitated to make the correct diagnosis. However, when the condition is encountered for the first time I doubt whether anyone would make the correct diagnosis. Here, the confusing element was the presence of a well-defined aortic insufficiency. The diagnosis was rupture of the aorta, dissecting aneurysm, relative aortic insufficiency due to distortion of the aortic ring, and rupture of the aneurysm into the pericardial sac.

The postmortem examination revealed a greatly enlarged heart, the left ventricle being much hypertrophied. The pericardial sac was filled with blood clot. All the valves were normal. The aorta showed marked arteriosclerosis but no evidence of syphilitic aortitis. At a point 7 cm. above the aortic ring there was a transverse tear through the intima of the aorta with the formation of a dissecting aneurysm, the dissection extending down to the aortic ring but distally only about 2 cm. Rupture of the aneurysm into the pericardial sac occurred near the reflection of the pericardium over the right auricle.

SUMMARY

Whereas it is our aim finally to arrive at an etiologic diagnosis of heart disease, it is seldom possible and always hazardous to make such a diagnosis directly. A more natural and more suc-

cessful method is to consider first the functional disturbances that are present, particularly those which have led to heart failure, then to predict the associated anatomic lesions, and from a consideration of these two and all other related factors to reach an etiologic diagnosis. Heart failure in all but very rare cases comes as a sequence to valvular disease, to hypertension, to congenital defects, to pericardial lesions, to pulmonary hypertension or to myocardial disease. A convenient method of procedure is first to decide in which of these six categories a case of heart failure belongs, and only then to attempt an etiologic diagnosis.

Cases of heart failure selected from clinicopathological conferences are presented to illustrate the operation of this method of diagnosis.

NAILING THE FRACTURED NECK OF THE FEMUR WITH THE AID OF THE FLUOROSCOPE

NEWTON C. BROWDER, M.D.*

BOSTON

MOST fractures of the femoral neck occur in feeble, old patients of sixty-five to seventy-five who are subject to hypostatic pneumonia, myocardial failure and serious bedsores. The method of treatment must combat these serious and frequently fatal complications by being simple, accurate and gentle enough to minimize the danger of shock, trauma, postoperative sepsis, and confinement to bed. The Smith-Petersen[8] and Sven-Johansson nailing operations,[7] with their numerous modifications, meet some of these requirements, but most of these procedures involve the risk of infection from a wide surgical wound, with considerable shock and loss of blood, and delay caused by taking numerous x-ray plates, which may prolong the anesthesia for one or two hours.

If a nailing operation is adopted, the technic should be simplified so that it will conform to the following standards:

The operation should not exceed thirty minutes from the administration of the anesthesia to the final closure.

The anesthesia should be of such small dosage that it offers no risk to the debilitated patient.

The direct visibility of the fluoroscope should be substituted for the indirect visibility of the x-ray plate. The fluoroscope saves time and is a more accurate and convenient method of guiding the nails, but only those of expert skill, who are doing such work frequently, should attempt it.

The skin incision needs to be only large enough to admit the nail, that is, 1 to 2 cm. long, thus preventing unnecessary loss of blood and loss of time involved in a larger incision.

No finger should enter the wound, and only a few special instruments are necessary, such as the Kirschner drill, guiding wires, nails, nail set, hammer, pliers and wire cutter.

IMMEDIATE TREATMENT

The patient is usually in some shock and considerable pain after the fall which fractured the hip. An injection of morphine and atropine will ease the pain and allow the surgeon to pull down the leg and rotate it inward, thus practically reducing the fracture. This will lessen the leg shortening, stop the hemorrhage and mitigate the shock, pain and muscle spasm.

While an assistant holds the leg in this position, traction straps of adhesive should be applied to both sides from the ankle to the trochanter with or without a Thomas splint, and a 5- to 8-lb. pull should be exerted on the straps to overcome the muscle spasm. This is essential to the success of the subsequent treatment.

As the patient will be fairly free from pain and will recover from shock in a few hours, the treatment of a decompensated heart or the control of the restlessness of an acute brain condition can be accomplished within the next few days. These patients usually become incontinent of urine or feces or both, and are very apt to develop bed sores within a few days. The ideal time for the operation is therefore on the second or third day after the fracture, although hips have been nailed successfully as long as six weeks after fractures.

OPERATION

We have discarded barbiturates and morphine in the preliminary anesthesia, believing that the respiratory depression caused by them may result in cardiac decompensation and pneumonia, especially in patients of over seventy. Spinal anesthesia is the most satisfactory form of anesthesia; 50 mg. of novocain and 5 mg. of Pontocain produce unconsciousness lasting thirty to forty minutes and give complete relaxation and absence of pain when the nail is driven in. In several cases where spinal anesthesia failed to work or more time was required than usual, thus allowing the effects of the spinal anesthesia to wear away, we gave the patient Pentothal Sodium or Evipal intravenously.

Local anesthesia has been used exclusively by

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Moore[4] with remarkable success. He injects 20 to 40 cc. of 2 per cent novocain intracapsularly and a small amount along the proposed skin incision. Our chief objection to local anesthesia is that it takes more time for it to work; it adds more fluid to the operative field, thereby increasing the danger of sepsis; it does not give complete muscular relaxation, particularly in patients who are operated on within a few days after injury; and the patient suffers pain when the nail is driven in. In fractures near the

Figure 1. *First maneuver in setting fracture.*

trochanter, where the novocain becomes disseminated outside the torn capsule, very little anesthesia is obtained.

Barbiturates alone, or in combination with morphine and scopolamin, have been used successfully with a few very elderly patients, but such anesthesia is not reliable and is extremely dangerous.

A surgeon, a scrubbed assistant, an assistant to hold the reduced leg, a nurse, an anesthetist and a roentgenologist are needed to perform the operation in the shortest time possible.

An anteroposterior x-ray film, taken on admission, will give the surgeon the location of the fracture. Many of the intertrochanteric fractures can be nailed if enough of the shaft remains intact to hold the nail.

Reduction of the fracture should be gentle, as very little force is required if traction straps have been applied for a few hours previously; more than one manipulation is seldom necessary. Frequent or violent manipulation increases the hemorrhage at the fracture site, injury to the soft parts and the subsequent shock. In several instances patients have died immediately after violent manipulations. We have employed a modification of Leadbetter's[3] method of reduction.

As soon as the spinal anesthesia produces relaxation the hip is reduced. An assistant grasps the iliac crest for countertraction. The surgeon stands at the outer side of the injured leg, grasps the front of the ankle with one hand, and bends the knee to right angles over his forearm (Fig. 1). He next flexes the hip to right angles, rotates it outward to disengage the broken ends of the fracture, and pulls directly upward in traction (Fig. 2). The fragments will be felt to

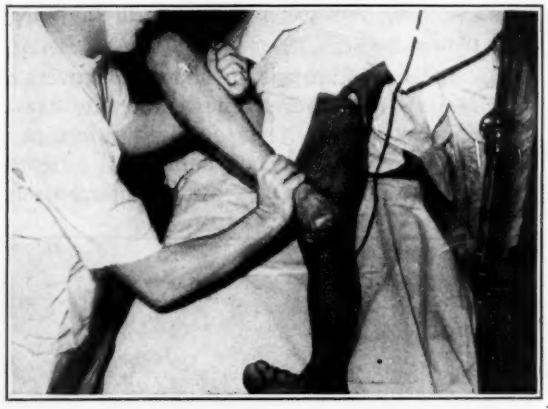

Figure 2. *Second maneuver in setting fracture.*

disengage. Held in this position, the leg is carried inward toward the other leg until the surgeon's hand on the ankle has passed over the other leg. With the traction sustained, the injured leg is gradually rotated inward and extended until it lies flat on the fluoroscopic table.

Circumduction of the leg, while held in traction, and internal rotation will free any part of the capsule that may have been caught between the fragments. The leg, held in constant traction and internal rotation (15 degrees), is then abducted 20 degrees (Fig. 3). If the fracture has not been

Figure 3. *Final position of the leg in the reduction of the fracture.*

reduced, the leg will not rotate inward the required 15 degrees from the neutral position. This failure is due to the fact that the surgeon did not keep constant traction during the reduction. The move-

ments in reduction should be very smooth, and require very little strength if the muscles are relaxed by the anesthesia. The leg is then held in constant traction by an assistant, internal rotation of 15 degrees and abduction of 20 degrees being maintained except when checking the lateral view. Failure to do this will often result in the distal fragments slipping upward, particularly in high fractures of the neck.

It is well to go through the above maneuvers on the uninjured leg several times before the day of the operation, so as to learn the slight difference in the arcs of motion peculiar to individual patients.

The reduction is checked by measurements for shortening and by fluoroscope. Both the roentgenologist and the surgeon should wear dark glasses so that there will be no loss of time for the accommodation of the eyes. If the reduction has been properly done, the angle of the neck will be a smooth arc without any depression or extension of fragments beyond this line. If the reduction is not perfect, the maneuver of reduction should be repeated. Only the anteroposterior view needs to be checked at this time.

Figure 4. *"Frog position" of leg for the lateral view of the hip either under the fluoroscope or by using an x-ray film.*

The hip region should be scrubbed with soap and water and alcohol and a dry sterile dressing applied just previous to the operation. Three per cent tincture of iodine is then applied from the crest of the ilium to the knee, and from the inner side of the thigh to its outer and under surface.

Stainless steel wires are inserted with a Kirschner wire drill. The use of 3/32-inch flat-headed wire drills with tapered points is preferable to that of the usual 2/32-inch drills.* The surgeon palpates the middle of the shaft of the femur on the outer side just below the great trochanter, as this is close to the middle of the anteroposterior diameter of the shaft and neck. Under the fluoroscope he locates the edges of the shadow of the acetabulum, which determine the exact center of the neck and head in the superior-inferior diameter. Without making any skin incision, and holding the drill parallel to the table top, he pierces the skin down to the bone just below the great trochanter in the middle of the shaft of the bone and drills the wire through the center of the neck and head to within 1 cm. of the cartilage surface of the head. He then drills a second wire within 1 cm. of the cartilage surface of the head. If the fluoroscope shows that either wire is not centered anteroposteriorly, it is well to remove it with pliers and insert another drill.

The position of the wires in the lateral view can now be quickly determined. The assistant, holding the traction on the leg, flexes the knee to right angles and the hip to 45 degrees, and rotates the hip outward so that the outer side of the heel is resting off the shin of the well leg (Fig. 4). This "frog position" of the leg has rotated the neck and head 90 degrees, giving a true lateral view of the position of the wires in the neck and head. Often it is found that one of the wires is too high anteriorly or too low posteriorly in the neck and head. If one of the wires is centered satisfactorily in the neck and head, the leg is replaced in its former position and the other wire is removed. If neither wire is centered in the lateral view, the one farther from the center is removed, the other wire remaining as a correction guide, and another wire is inserted so as to be centered in both the anteroposterior and lateral views. Even if many trials are needed to get a wire in the axis of the neck and head, this should be accomplished before the next step is taken. It is much more difficult, time-consuming and shocking to the patient to attempt to change the direction of the nail than that of the wire. The wire-drilling causes little harm and the multiple drill holes aid union.

Female patients require nails varying in length from 8.5 to 9.5 cm. Those for male patients vary

*The larger drills do not bend so easily as the small ones when the lateral view is checked in the frog position.

from 9 to 11 cm. Johansson nails come in 0.5-cm. lengths, and nails of the approximate sizes required should be on the sterile table. The exact size of the nail desired is quickly and easily determined. A drill wire the same length as the one in the bone is pushed down to the bone through the same opening (Fig. 5). This wire will protrude beyond the first by the same distance as the length of the first wire in the bone. A nail slightly shorter than this measurement should be used, so as to allow for the impaction at the fracture site, which always shortens the distance by about 0.5 cm. A nail thus chosen will

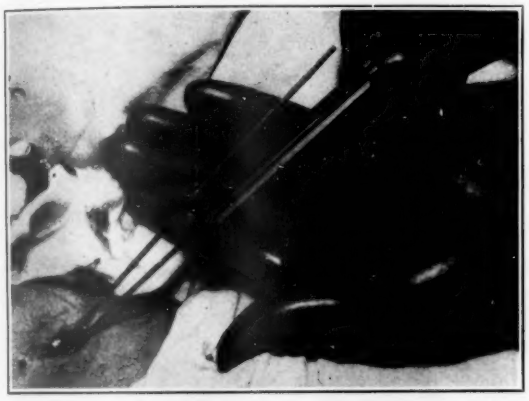

Figure 5. *Measurement of the nail.*

fit snugly against the cortical edge of the femur without penetrating the cartilage of the head.

With a Bard-Parker knife a 2-cm. incision is made in the skin at the site of the wire. The point of the knife is pushed down to the periosteum on both sides of the wire. This allows a free passage of the 1-cm. head of the nail through the soft tissues.

The nail is threaded on the wire guide and pushed with the thumb down to the bone. A cylindrical nail set 1 cm. in diameter and 10 to 15 cm. long, with a head 2.5 cm. in diameter and a channel bored through its axis, is then threaded on the wire external to the nail. The nail set will transmit the blow of the hammer to the head of the nail without the necessity of enlarging the skin incision (Fig. 6). When the nail has penetrated the neck of the femur by about 5 cm., the wire may protrude beyond the nail set; if so, 5 cm. of the wire should be cut off with pliers. The nail set is replaced on the wire and the nail is driven into the head until it is within 1 cm. of the cartilage surface as shown by the fluoroscope.

A Kelly snap should always be placed against the head of the nail for counterpressure before pulling the wire out. If the wire is slightly bent, it may jam in the nail, so that great force is necessary to remove it, and the nail will come out of the bone with the wire. Usually, however, by turning the wire drill counterclockwise a few turns it can be removed with little or no force.

Only one stitch should be taken, as this will leave a slight gap in the small wound through which any blood or serum may drain into the dressing. This helps to prevent a sterile abscess around the nail, which is sure to loosen it, and to allow it to work outward so that it will no longer hold the fragments of the fracture together.

Anteroposterior and lateral x-rays should be taken immediately after the operation as a matter of record (Figs. 7 and 8). Lateral views taken with the leg in the "frog position" should not be repeated for several months, as this position tends to

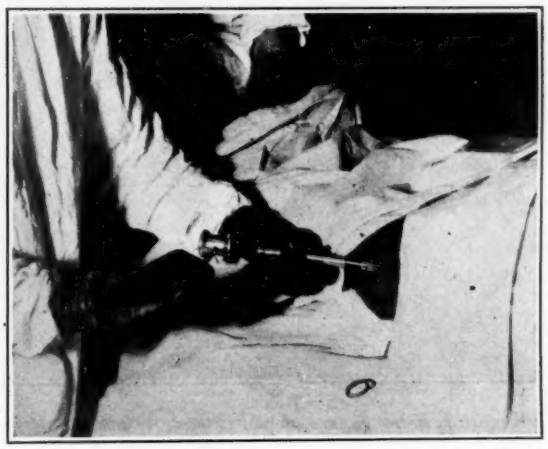

Figure 6. *Nail and nail set in place; driving the nail through the small incision.*

loosen the nail. Anteroposterior views should be photographed every month. If the patient develops pain in the hip, an x-ray check-up should be made.

Postoperative treatment is essential to successful results. The patient may move the leg on the following day and be allowed to sit out of bed in three to five days if the pain in the hip has subsided. As the neck has only the thickness of an eggshell it is advisable to apply an ambulatory Thomas splint for weight-bearing during the next four to six months. Crutches should be used during the first eight to twelve weeks, and again when the Thomas splint is removed until the eighth or tenth month.

RESULTS

From June, 1935, to April, 1938, a total of 53 cases was operated on at the Boston City Hospital according to the technic described above. Nineteen of the patients were men and 34 were women. The fractures were classified as follows: intertrochanteric, 12; low-neck, 10; mid-neck, 20;

high-neck, 11. Spinal anesthesia was used in 47 cases, and local anesthesia in 6. The Johansson type of nail was employed in 45 cases, that of Magnusson or Böhler in 3, and Moore's pins in 5.

Perfect reduction was obtained in 49 cases, and of the remainder 2 showed a varus and 2 a valgus deformity. Postoperative fixation was accomplished in 30 cases by the use of a Thomas splint, and in 12 cases of intertrochanteric fracture by

Figure 7. *X-ray picture of nail in place — anteroposterior view.*

means of 5-lb. traction applied for from two to four weeks. The 39 patients who survived operation and who were mentally sound were up and about on crutches on an average of 17 days after operation, and the average stay in the hospital excluding those patients who died in the hospital or who were still there on April 1, 1938, was 37.6 days. Full weight-bearing was possible after an average

Table 1. *End Results of Operation.*

END RESULT	NO. OF CASES	PERCENTAGE OF TOTAL
Nail slipped in	11	21
Complete union	20	38
Fibrous union	8	15
Non-union	15	28
Shortening	19	36
Absorbed neck	10	19
Necrosed head	1	2

postoperative period of 9.4 months in 11 cases (21 per cent), and weight-bearing supplemented by the use of crutches was obtained in 29 (55 per cent); of the remainder, 11 patients died postoperatively and 2 were incapable of weight-bearing because of their mental condition.

The end results of the operation are listed in Table 1. Of the 42 patients surviving operation excellent functional results were obtained in 24, fair results in 13 and poor results in 5.

Postoperative complications occurred in 30 cases (57 per cent) and are listed in Table 2. By far the majority of these are the accompaniments of old age. Mild sepsis about the nail was observed in only 2 cases.

Eleven patients with an average age of 72 years died from two to fifty-two days after operation, a mortality of 21 per cent. Three of these patients were operated on for the relief of pain with no

Figure 8. *X-ray picture of nail in place — lateral view.*

expectation of recovery, and there were several others in whom the condition of the cardiorespiratory or genitourinary system was such that little hope was entertained for their survival. The 11

Table 2. *Postoperative Complications.*

POSTOPERATIVE COMPLICATION	NO. OF CASES	PERCENTAGE OF TOTAL
Bronchopneumonia	10	19
Chronic myocarditis with decompensation	7	13
Chronic myocarditis and auricular fibrillation	2	4
Decubitus of severe type	7	13
Mental psychosis in acute attack	6	11
Alcoholic with delirium tremens	3	5
Arteriosclerosis	6	11
Sepsis about the nail	2	4
Acute infectious arthritis	2	4
Cirrhosis of liver	1	2
Atelectasis	1	2
Pulmonary embolism	1	2
Cerebral thrombosis	1	2
Anemia	1	2
Flexion deformity of knees and hips	1	2
Herpes zoster of supraorbital nerve	1	2
Hyperthyroidism	1	2

immediately fatal cases are listed in the upper part of Table 3. Five other cases, listed in the

lower part of the table, died from four months to one year following operation. Since the causes of death in the latter were not directly related to operation, the cases are not included in figuring the operative mortality.

Table 3. *Fatal Cases.*

CASE	AGE	DIED POSTOPERATIVE	CAUSE OF DEATH
	yr.	*days*	
J. B.	65	13	Chronic myocarditis with decompensation
C. B.	67	5	Chronic myocarditis with decompensation (acute alcoholic)
P. C.	73	2	Chronic myocarditis with decompensation
M. B.	89	36	Chronic myocarditis with decompensation
J. M.	64	10	Chronic myocarditis with decompensation (delirium tremens)
S. C.	68	52	Bronchopneumonia (decubitus; arteriosclerosis)
R. J.	67	7	Bronchopneumonia (chronic myocarditis; auricular fibrillation)
E. Mc.	76	3	Bronchopneumonia (arteriosclerosis)
S. P.	85	11	Bronchopneumonia
E. C.	62	31	Bronchopneumonia
E. M.	72	8	Pulmonary embolus
		mo.	
M. N.	84	4	Hypostatic pneumonia
J. M.	70	7	Cause unknown (hypertension; pulmonary tuberculosis; empyema)
M. C.	73	7	Bronchopneumonia (cerebral thrombosis; arteriosclerosis)
E. P.	80	7	Cause unknown
A. B.	72	12	Cancer of the tongue

SUMMARY

1. A method of setting and nailing the fractured necks of femurs is described which is quick and accurate because of its visibility under the fluoroscope. It requires only twenty to thirty minutes after the induction of a minimum amount of spinal anesthesia. The 3/32-inch stainless steel wires, 22 cm. in length, with flat-headed, tapered drill points, are strong enough to fix the fracture and permit fluoroscopic observation of the lateral view. The exact length of the desired nail can be measured quickly and accurately after the guide wire has been drilled into the neck and head. A second wire of equal length is pushed through the same opening parallel to it until it strikes the bone. It will protrude beyond the first wire the exact length that the first wire has entered into the bone. A nail slightly shorter than this length is chosen.

The results of the application of this method in 53 consecutive cases are given.

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ANAL FISSURE

An Evaluation of Treatment With Oil Injections

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IN 1927 Yeomans and his associates[1] introduced the use of injection of anesthetics in oil in the treatment of pruritus ani. Their suggestions were later used by Gabriel[2] in the treatment of anal fissure. This new treatment has gained much favor and a mass of literature concerning its results has accumulated. In the past two and a half years 129 patients with anal fissure have been seen in the Rectal Clinic of the Out Patient Department of the Massachusetts General Hospital. These constitute 15 per cent of all patients seen there. Of these cases, 76 were treated before the introduction of oil treatment; the others, 53, were treated with oil injection.

The important anatomic landmarks to bear in mind in the consideration of a fissure are the anal canal, the internal sphincter, the crypts of Morgagni and the small fibrous papillae between the crypts. The anal canal is 2 to 3 cm. long and is surrounded by the internal sphincter, with the external sphincter encircling the orifice. The crypts and the papillae make up the dentate line which forms the innermost limit of the anal canal. The crypts, eight to ten in number, are usually only shallow depressions 1 to 2 mm. deep. Occasionally, however, we find crypts measuring 1 cm. or more in depth, simulating diverticula, at the mucocutaneous junction; we consider the latter to be important in the pathogenesis of fissures and fistulas. The anal canal is lined with squamous epithelium and contains sensory nerves, whereas the mucous membrane of the rectum beyond the dentate line does not.

A fissure is a chronic ulceration situated in the middle or lower third of the anal canal, varying in diameter from a few millimeters to a centimeter. It is anterior in only 10 per cent of cases, and is rarely lateral. In a well-established chronic case there are two sentinel piles: the internal, which represents fibrous enlargement of the normal papilla at the orifice of the crypt, and the external, which is at the margin of the anal orifice and is always visible outside the anus. The ulceration lies between these. The diameter of the sentinels varies with the chronicity and activity of the fissure, being from a few millimeters to several centimeters. The ulcer is surrounded on all sides by scar tissue firmly adherent to the external sphincter. In 61 per cent of our cases the presence of sentinel piles was noted, and in 39 per cent deep crypts were observed.

The cause of fissures has been variously explained as injury to the anal canal from passage of hard fecal masses, as infection of a crypt and subsequent sloughing, and as the tearing of a crypt and subsequent infection. We regard the infection of a deep crypt and either spontaneous ulceration of the thin wall or its tearing by passage of hard feces as the causes of this condition. We have frequently found crypts more than 1 cm. long in an otherwise normal anal canal. These deep crypts act as diverticula, becoming infected because of stasis and inadequate drainage. If the lumen of the infected crypt is directed downward along the wall of the anal canal between the mucosa and the sphincters, the subsequent sloughing of the wall will result in a fissure; on the other hand, if the lumen of the crypt is directed away from the anal canal through the fibers of the internal sphincter, the infection, breaking through the blind end of the pocket, enters the tissues of the ischiorectal fossa and forms a perianal or ischiorectal abscess with resulting anal fistula.

After ulceration of the wall of the infected crypt, healing may take place rapidly provided there is sufficient drainage, and no chronic fissure results. The crypt may not ulcerate at all, the infection gradually subsiding after one or two weeks of acute cryptitis, to recur at some future date. Usually only a part of the crypt wall ulcerates, leaving inadequately draining sinuses at the upper and lower ends. These keep up the infection; scar tissue forms that distorts the original picture; the papilla hypertrophies at the orifice of the crypt and gives rise to the internal sentinel, while the skin at the anal margin forms the external sentinel. The inflammation extends to the external sphincter with resulting spasm, and the picture of a chronic fissure is then complete.

In our series there were 69 women and 60 men. The greatest incidence was between the ages of forty and fifty, as shown in Table 1. The average age was forty-one.

The commonest symptom was pain. This was present in 96 per cent of our cases. It was very

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marked at the time of bowel movement and gradually subsided. Four per cent of cases had no pain whatever. About half the patients noticed bleeding, which was usually slight. Other occasional symptoms were itching, constipation, anal discharge, the presence of a lump, tightness of the sphincter and tenesmus. The duration of symptoms varied from a few days to many years. Some

Table 1. *Age Incidence.*

AGE IN YEARS	NO. OF CASES	PER CENT
1-10	1 (age 6 yr.)	1
10-20	0	0
20-30	26	20
30-40	34	26
40-50	37	28
50-60	19	15
60-70	8	6
70-80	4	3
Total	129	

patients had had repeated attacks. Twenty patients came to the clinic less than one month after onset and 24 came from one to three months afterward. The duration of symptoms for 88 cases is given in Table 2.

Table 2. *Duration of Symptoms.*

DURATION OF SYMPTOMS	NO. OF CASES	PER CENT
1 mo. or less	20	23
1 to 3 mo.	24	27
3 to 6 mo.	14	16
6 to 12 mo.	8	9
1 to 10 yr.	14	16
10 to 20 yr.	3	3
Many years	5	6
Total	88	

In 113 patients (88 per cent) the fissure was posterior and in 16 anterior. In the latter group there were 11 women and only 5 men.

We place the patient in the right Sims position. The skin about the anus is prepared as it is for an operation. One per cent novocain is injected subcutaneously through a 26-gauge, 4-cm. needle, beginning about 2 cm. from the posterior commissure. This infiltration is carried out fanwise and may be extended into the external sphincter. The injection of novocain may be dispensed with, since that of the oil causes only slight discomfort. With the left index finger in the anus as a guide, we inject the oil solution with a Luer Lok syringe through a 24- or 25-gauge needle. This requires considerable force, and the oil goes in slowly, but there is less danger of slough. Injection is continued as the needle approaches the external sphincter, and the solution is injected into the muscle and under the ulcer. The amount used varies from 1 to 5 cc. The treatment may be repeated at weekly intervals. Our cases received from one to four injections.

After the injection the patient usually has immediate relief of pain. There are complications, however, that must be considered. Two of our patients developed small abscesses at the site of injection one week after treatment. These were opened, with subsequent healing of the fissure. One patient had a pinhead-sized area of skin necrosis. Another developed a small, superficial fistula.

Two solutions have been used for injection. Gabriel's modified solution is oil of sweet almonds containing 0.5 per cent Nupercaine, 1.0 per cent phenol and 10.0 per cent benzyl alcohol. Morgan's solution[3] is peach-kernel oil containing 1.5 per cent procaine, 6.0 per cent Butesin and 5.0 per cent benzyl alcohol.

The end results on outpatients are sometimes difficult to obtain. We depended entirely on the voluntary return of the patient for observation.

Table 3. *End Results of Treatment with Oil Injection.*

	NO. OF CASES	RELIEVED		RECURRED	
		no.	%	*no.*	%
Oil injection only	39	25	64	14	36
Oil injection and minor operation	11	6	54	5	46
	50	31	62	19	38

Some patients failed to return after receiving treatment or advice, and others did not return after operation. No doubt many of these were relieved; others perhaps went to other clinics. In determining the results of treatment we were able to follow 95 cases for various periods up to one year.

As shown in Table 3, in 39 cases oil alone was used. In 36 per cent there was a recurrence, after an average of three months. In 11 cases a minor operation* was done in the clinic under oil anesthesia and in 46 per cent of these there was a recurrence. Of all cases of oil injection followed up, 62 per cent were relieved and 38 per cent had recurrences after three months. The results of treatment without oil injection are shown in Table 4. Again we have eliminated all cases not

Table 4. *End Results of Treatment without Oil Injection.*

	NO. OF CASES	RELIEVED		RECURRED	
		no.	%	*no.*	%
Operation	35	29	83	6	17
Miscellaneous treatment	10	9	90	1	10
	45	38	84	7	16

followed. Eighty-three per cent of 35 patients were relieved by operation, in comparison with 64 per cent relieved by oil injection; and 90 per cent of 10 patients gained relief from various kinds of treatment—mineral oil, sitz baths and cauterization with silver nitrate. Most of the latter were cases of small fissure without deep crypts

*Removal of external sentinel pile to allow better drainage.

and sentinel piles, but many of the patients receiving oil injection also had small fissures and would probably have been cured with moist heat and bowel regulation.

The patients listed above were all ambulatory. The number of operations in the hospital has been reduced since the introduction of oil injection, as shown in Table 5.

Table 5. *Treatment of Anal Fissure Before and After Introduction of Oil Injection.*

	1935		1937	
	NO. OF CASES	PER CENT	NO. OF CASES	PER CENT
House operation	20	46	12	17
Refused operation	8	20	2	3
O.P.D. minor operation	4	10	0	0
Bowel regulation and moist heat	11	24	6	10
Oil injection	0	0	24	42
Oil injection and O.P.D. minor operation	0	0	16	28
	43		60	

The main feature in the oil-injection treatment of anal fissure is prolonged anesthesia with resulting relaxation of the sphincter, pain thus being relieved and freer drainage established. In most cases the pain disappears before the patient leaves the examining table. Whereas before the injection digital or instrumental examination may be impossible, immediately afterward it can be performed without pain. The anesthesia lasts from one to three weeks, gradually wearing off. In the meantime the salutary effects outlined above allow healing and keep the patient symptom-free. In most cases, especially those patients having simpler fissures without large sentinel piles and underlying pockets, healing takes place before complete sensation returns. This happened in 64 per cent of our injected cases. The simpler cases, however, can usually be cured by a regimen of bowel regulation and moist heat applications. Before we started the oil injections we cured 20 per cent of all cases by this simple method. In well-established fissures, with two sentinel piles, scar tissue and underlying deep crypts, oil injections give fully as much relief as in the simple fissures, and the ulcer may even heal up; but the presence of inadequately draining sinuses maintains the infection, and sooner or later, when anesthesia has disappeared, the vicious circle of pain and spasm returns with recurrence of the fissure. This happened in 38 per cent of our injected cases. The percentage of cases relieved by oil injection is somewhere between 40 and 60 of those treated by this method. The avoidance of a hospital stay, rather than superior results, justifies the use of this method as against operation, which can be performed later on if the oil injections fail.

CONCLUSION

Oil injections in the treatment of anal fissure give the patient immediate relief from pain. In many cases the ulcer may heal without further treatment. In well-developed chronic cases, however, it does not take the place of operation.

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THE SPIVACK GASTROSTOMY

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GASTROSTOMY was one of the first operations performed by surgeons on the abdominal cavity. Sédillot[1] was the first to perform the operation on a human being in 1849; the patient died of peritonitis. During the next twenty-five years there were many unsuccessful attempts, until Jones[2] performed the first known successful gastrostomy in 1875 (quoted by Spivack[3]).

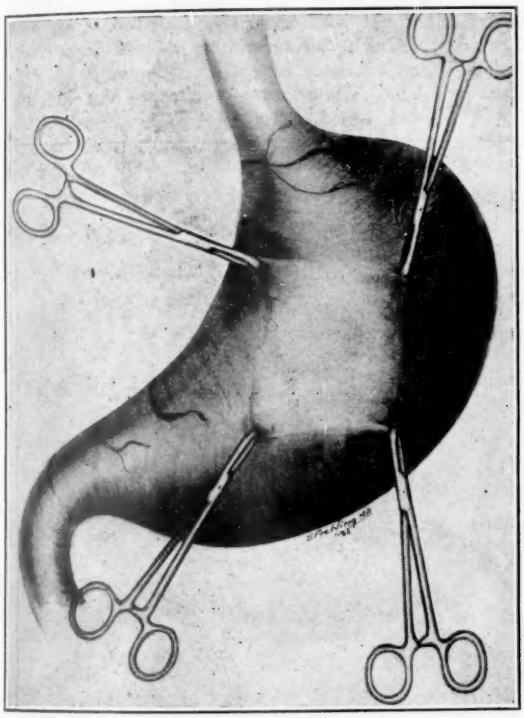

Figure 1. *Area 7.5 by 5.0 cm. selected on the anterior wall of the stomach.‡*

Throughout the history of gastrostomy the problem presented to the surgeon has been the back flow of the gastric contents through the fistula produced by the operation, and all attempts at technical improvement have been directed toward preventing the leakage. Various methods have been devised, all of which are ingenious in principle but fail in practice.

Under the Hahn[4] method, a cone was constructed from a part of the stomach, various devices being used for compression of the cone. This method has long since been discarded. Von Hacker[5] and Gerard[6] attempted to make a sphincter out of the abdominal wall. Ullmann[7] tried to construct a sphincter in the stomach wall. All these attempts proved unsuccessful.

The methods in which a canal lined with serosa is made in the anterior gastric wall include those of Stamm[8] and Witzel;[9] they are the procedures in commonest use today. Their advantages are that the operations can be done simply and that the canals serve efficiently where only a temporary gastrostomy is needed. In addition to the disadvantage which they possess in common with the other methods mentioned, they have the particular one of a tendency to obliteration because the tube is lined with serosa.

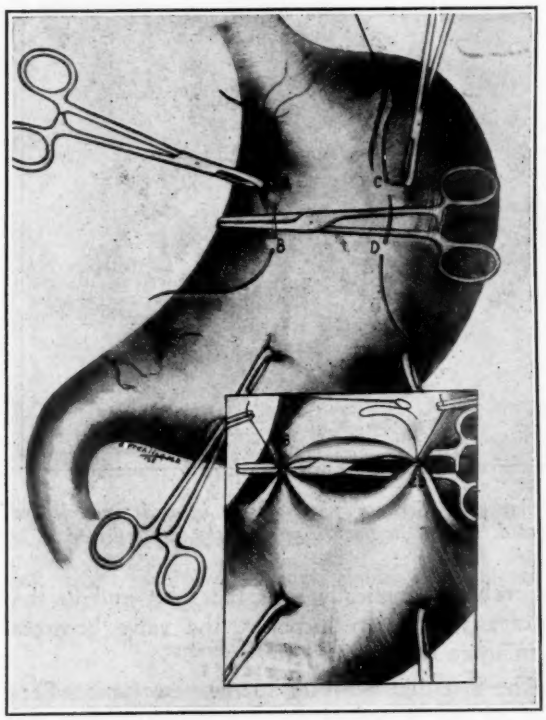

Figure 2. *Sutures taken through A and B, and C and D, over artery forceps. Insert: sutures tied at AB and CD, so doubling the anterior gastric wall.*

Under the methods of Marwedel[10] and Schnitzler,[11] a canal is made in the portion of the anterior gastric wall not lined with serosa, in an attempt

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‡All the illustrations are adapted from those which appear in Dr. Spivack's book *The Surgical Technic of Abdominal Operations* and are reproduced through the courtesy of the publisher, S. B. Debour, Chicago, Illinois.

to overcome obliteration. This procedure does not meet with success in practice. The construction of a mucosa-lined tube, using the anterior wall of the stomach, distinguishes the methods of Janeway,[12] Depage,[13] Beck and Carrell[14] and Hirsch.[15] Here again there is no way of preventing the gastric contents from leaking back through the fistula.

Spivack[16] in 1929 described a method in which a valve is constructed from the stomach wall, situated at the base of a tube, also made from the stomach wall. The structure is so designed that

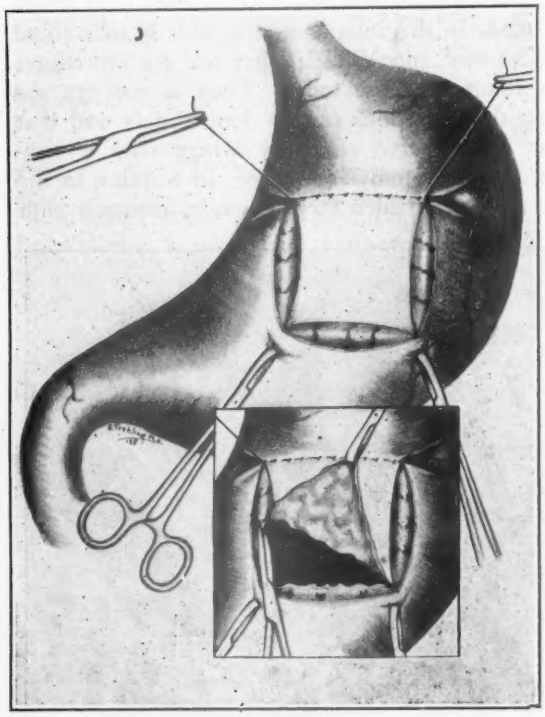

Figure 3. *Seromuscular incision is made; vessels are ligated. Insert: stomach is opened, the flap reflected.*

the valve hermetically seals the tube, and as the intragastric tension increases, the valve becomes even more watertight.

The first case admitted to the new Cancer Section of the Westfield State Sanatorium was the subject for a Spivack gastrostomy, and was listed first on the operating-room register. Aside from its possible historical interest, we believe that the case and a description of the operation deserve publication, because of the apparent lack of familiarity of New England surgeons with this method. From our experience with dogs and with the case presented, it is our opinion that the method is the one of choice and is worthy of wider practice.

CASE REPORT

P. S., a 74-year-old man, was admitted to the hospital on December 6, 1937. For the previous 6 weeks he had had increasing difficulty in swallowing even liquids in small amounts. There was a weight loss of 30 lb., associated with increasing weakness. Thirty-six hours before entry the patient was unable to void for the first time and required repeated catheterizations.

Physical examination showed an emaciated elderly man with wasting of the subcutaneous tissues, which were in a state of dehydration. Advanced peripheral arteriosclerosis was present. The bladder was distended. The prostate was moderately enlarged and boggy in consistence, and in the lower pole of the right lobe there was a discrete, firm nodule about 1 cm. in diameter. On admission the red-cell count was 2,200,000, and the hemoglobin 59 per cent (Sahli). Urine samples showed slight traces of albumin, and variable numbers of white and red cells in the sediment. The nonprotein nitrogen was 20.6 mg.

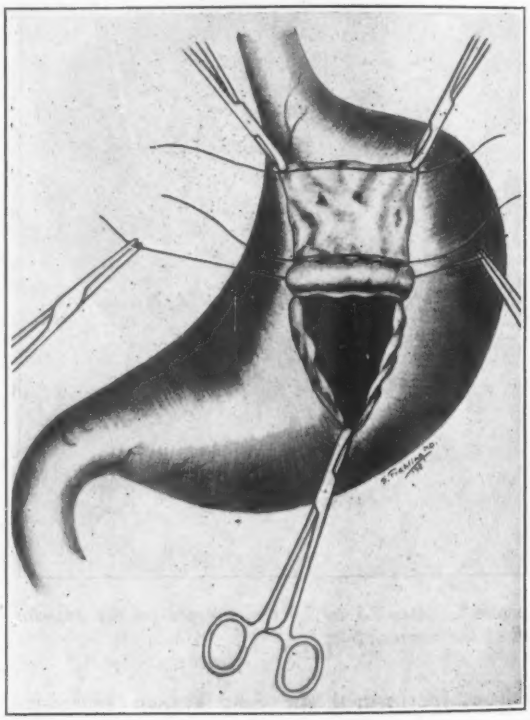

Figure 4. *Flap is completely reflected, and the valve is exposed. Sutures are taken through the corners of the flap and construction of a tube is begun.*

per cent. A blood Hinton test was negative. X-ray examination of the chest was negative. The esophagus after ingestion of barium showed a marked filling defect 5 cm. in length at the junction of the upper and middle thirds, having the appearance of a neoplasm. A plain abdominal film revealed gallstones and hypertrophic changes in the lumbar spine. An intravenous pyelogram showed slight dilatation of the pelvis.

The patient continued to have urinary retention after entry, necessitating constant bladder drainage by an inlying urethral catheter. Bilateral vasectomy was done to prevent a descending infection. The water balance was reestablished in about 1 week, and 10 days after admission

a gastrostomy was performed under local anesthesia. The postoperative course continued uneventful so long as adequate bladder drainage was maintained. Feeding through the gastrostomy was begun 4 hours after operation, and in three weeks the diet was gradually increased to 2500 calories every 24 hours. The patient was out of bed on the 8th postoperative day. There was never any leakage or reflux of feedings from the gastrostomy.

Figure 5. *Corners of flap tied around artery forceps forming the tube. Insert: closure of opening in stomach begun.*

Esophagoscopy, done during the 4th week, demonstrated a carcinoma in the middle third, with constriction, bleeding and solidity. A biopsy showed epidermoid carcinoma. Several days later the patient developed intravesical bleeding, which increased in spite of frequent bladder washings, and it was necessary to relieve great bladder distention by the removal of clots by vigorous suction through the cystoscope. Circulatory collapse ensued, requiring glucose infusions and blood transfusion. During the patient's shock there was acute gastric dilatation, aspirations producing 32, 14 and 12 oz. on three occasions. It is interesting to note that in spite of the large amount of stasis in the stomach, apparently under great tension, there was no leakage from the fistula. The patient did not recover from the circulatory failure and died 3 days after the onset of the bladder hemorrhage, 35 days following the gastrostomy.

Postmortem examination revealed 50 cc. of serosanguineous fluid in the left pleural cavity and 500 cc. of straw-colored fluid in the right pleural space. The lower lobe of the left lung was hemorrhagic. In the pulmonary artery leading to the left lower lobe was a recent antemortem thrombus slightly adherent to the intima. In the esophagus at the level of the arch of the aorta was an ulcerated area with a slightly elevated and convoluted border, extending around the entire circumference and measuring 5.5 cm. in length and 3.0 cm. in width, causing marked constriction of the esophagus. On section the tumor was seen to extend and infiltrate the periesophageal tissue and the trachea. The opening of the gastrostomy from within the stomach was found with difficulty; the mucosa surrounding it and forming a valve (Fig. 9*b*) was rather firm in consistence. The liver, spleen and pancreas were negative. Minute abscesses were seen throughout the edematous and friable kidney parenchyma. The mucosa of the bladder was congested, and a hemorrhagic area was present on the trigone, where the mucosa was apparently denuded by the catheter. The prostate was diffusely enlarged and composed of homogeneous tissue. No gross evidence of carcinoma was seen in the prostate. The anatomical diagnoses were: carcinoma of the esophagus, acute pyelonephritis, chronic cystitis, left pulmonary thrombus, left hemothorax and right hydrothorax.

Figure 6. *Opening in stomach and tube completely closed. Insert: seromuscular suture layer begun.*

DESCRIPTION OF OPERATION

The abdomen is opened through an upper midline incision, and the stomach is exposed in the usual manner. A quadrangular area about 5 cm. wide and 7 cm. long is selected on its anterior surface. In more contracted stomachs, the area may be shorter, 6 cm. long. An Allis forceps is placed at each corner of the selected quadrangle, as shown in Figure 1. The flap may be made in any direction, although Spivack's description indicates a reversed "U."

A seromuscular suture is taken between points AB and between CD (Fig. 2), the distance be-

tween them being from 2 to 4 cm. The ends of the threads are tied and clamped. The step is carried out over an artery forceps, and closure of the apposing serosa is completed with a running seromuscular stitch, using one end of the suture connecting C and D, and the upper half of the

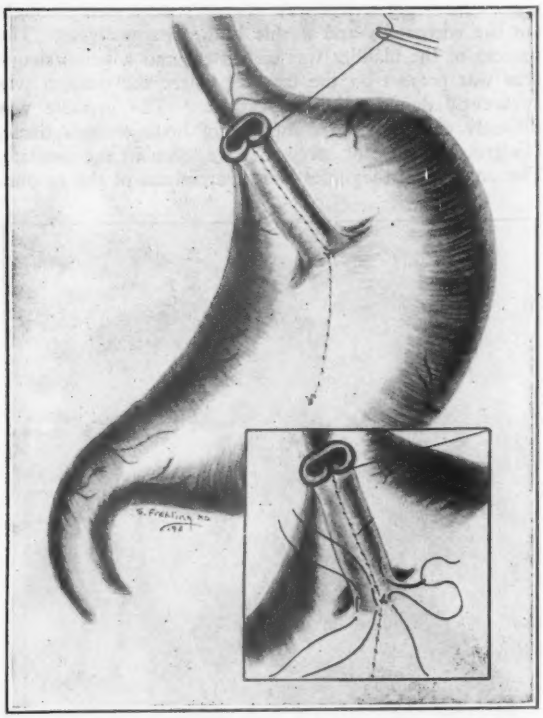

Figure 7. *Stomach and tube completely re-enforced by seromuscular suture. Insert: making seromuscular re-enforcing layer or cuff around base of tube.*

flap is thus doubled. The artery forceps lying behind the bridge may then be withdrawn.

Points AB and CD are connected with the lower corners of the quadrangle by two vertical seromuscular incisions, and the two lower angles are connected by a transverse seromuscular incision (Fig. 3).

The vessels and the mucosa are thus exposed, and the blood vessels are ligated. The mucosa is incised, and the flap produced is reflected (Fig. 4), exposing at its base the valve effected by doubling the seromuscular layer in the earlier step. Two sutures are then taken, one through the upper corners of the flap and the other through the two corners at its base (Fig. 4). The two suture ends of the seromuscular suture forming the valve, which have been held for guy sutures, are cut short, and the ends of the sutures through the upper and lower corners of the flap are tied and clamped (Fig. 5).

An artery forceps is inserted through the tube thus formed, and the opening in the stomach is closed with an over-and-over suture (Fig. 5, insert). The lips of the tube are closed over the forceps, also by an over-and-over suture (Fig. 6). After the first layer is completed a re-enforcing seromuscular

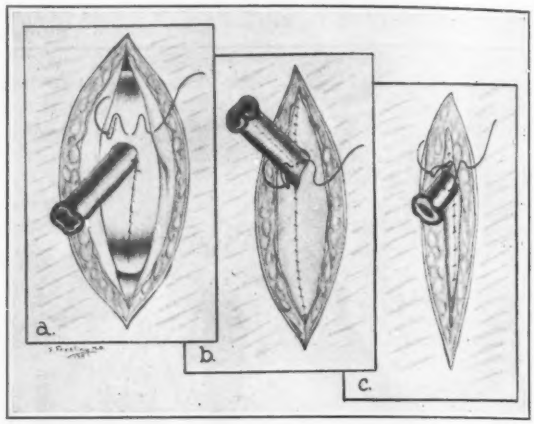

Figure 8. *Supporting stomach and tube in closure of abdomen: (a) suturing stomach to peritoneum; (b) suturing anterior sheath to tube; and (c) suturing subcuticular layer to tube.*

suture is made (Fig. 6, insert; Fig. 7). Three interrupted seromuscular sutures are taken for re-enforcement at the weak spot at the junction of the tube with the stomach (Fig. 8, insert).

The anterior wall of the stomach is sutured to the peritoneum (Fig. 8*a*). A suture is taken

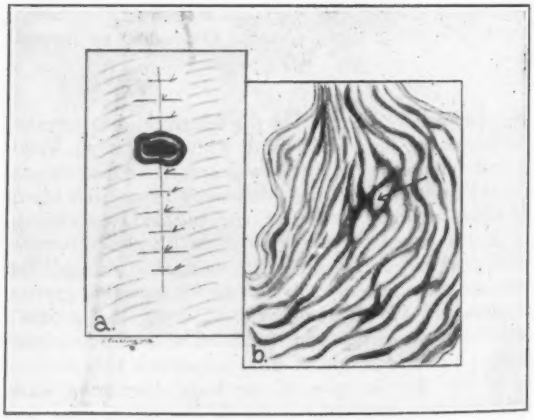

Figure 9. *Final view: (a) mucosa forms a cuff over portion of tube above surface of skin (serosa not exposed); and (b) view of stomach interior — valvular flap seals internal opening of tube.*

through the peritoneum and the posterior rectus sheath, then through the seromuscular layer of the stomach at a point just below and proximal to the base of the tube, and then through the other lip of the peritoneum and the transversalis fascia.

A similar suture is taken at a point just below and distal to the base of the tube. Before tying these sutures, care should be taken that the tube is not constricted.

After the peritoneum has been closed in the usual manner, the tube is sutured to the fascia (Fig. 8*b*) as in the previous step. The tube is attached to the skin in four places to fix the structure completely. Sutures are taken through the subcuticular layer of the skin and through the seromuscular layer of the tube about 1 cm. from the open end (Fig. 8*c*). Mucosa covers the portion of the tube exposed above the skin surface (Fig. 9*a*).

When the stomach is contracted and very small, as is frequently the case with an obstructed esophagus, Spivack[16] utilizes the posterior wall for making the tubovalvular gastrostomy. His method may be used for a temporary procedure, since when its need ceases the extraperitoneally situated tube can be resected very simply under local anesthesia.

CONCLUSIONS

In this method we have the perfection that has been sought throughout the history of gastrostomy. It eliminates two major difficulties which always confronted surgeons: the canal does not tend to obliterate and the stomach remains watertight. During an episode of acute gastric dilatation with the case reported, as much as 32 oz. of stomach content was removed at one time and no leakage from the gastrostomy occurred. We consider the tubovalvular gastrostomy of Spivack as the method of choice.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24351

PRESENTATION OF CASE

A sixty-four-year-old, white, Canadian nurse entered the hospital with the complaint of numbness of the lower extremities.

Ten months before entry she began to notice numbness of both feet, not associated with tingling, loss of position sense, pain or disability. Although her feet had always had a tendency to be cold, at that time they became intensely cold and felt lifeless and heavy. During the ten months before entry this feeling of numbness gradually extended up her legs and trunk until it reached the level of her breasts. The anesthesia became so complete that at times she was unable to tell whether her legs were covered with bedclothes. She also had some hypersensitivity and pruritus in the area of numbness, so that she was unable to wear a corset. For about five months before entry she was somewhat unsteady on her feet both day and night. On two occasions she fell to the floor after suddenly standing up. She had had a cataract removed from her right eye four years before entry, and the vision of her left eye was quite poor. She was unable to say whether the unsteadiness of her gait was due to her poor vision or to her present illness. About four months before entry she began to drag her right foot, and some time after that she began to have involuntary twitching movements of her legs. She never had pain in her legs. About five months before entry she had an attack of severe pain in the region of the vagina which was relieved by urination. She had no other urinary symptoms. About four months before entry, while lifting an invalid whom she was attending, she suddenly felt a severe tearing pain starting in the region of the twelfth left rib in the posterior axillary line and radiating around to the front and down toward her leg. The pain was of brief duration, but soreness persisted in the local area, and she stated that a large swelling appeared there which gradually subsided.

Her past history and family history were essentially negative. She had had no symptoms of tuberculosis and had not been exposed to it for at least twenty years. She had had no recent weight loss.

Physical examination revealed a well-developed and nourished woman in no apparent discomfort. The heart and lungs were negative. The blood pressure was 190 systolic, 100 diastolic. The upper border of the liver was percussed at about the sixth interspace, and the edge was palpable about three fingerbreadths below the costal margin on deep inspiration. The abdomen was otherwise negative. The fundus of the right eye showed pigmentation in the macular region, and just temporal to this were two, small, grayish cystic areas invaded by small blood vessels and surrounded by fresh hemorrhages. The visual fields were normal. Except for diminished hearing the cranial nerves were normal. The gait was somewhat unsteady, and there were involuntary twitching movements of the legs, especially on the right. A Romberg sign was present. There was diminished strength in both legs and slight atrophy of the right. The deep tendon reflexes of the legs were equal and hyperactive. There was a positive Babinski sign on the left, and an equivocal plantar reflex on the right. The abdominal reflexes were absent. Examination of the arms was negative. All forms of sensation were diminished below the level of the eighth dorsal vertebra, and there were scattered areas of paresthesia.

The temperature was 99°F., the pulse 85. The respirations were 20.

The urine examination was negative. It contained no Bence-Jones protein. The blood showed a red-cell count of 4,290,000 with 75 per cent hemoglobin and a white-cell count of 6300 with 64 per cent polymorphonuclears. The guaiac test on the stool was negative. The bleeding time was 1 minute, the sedimentation rate was 0.45 mm. per minute, and a 1:100 tuberculin test was positive, although a 1:500 test was negative. The serum protein was 7.1 gm. per cent. A blood Hinton test was negative. A cystometrogram showed a hypotonic curve.

An x-ray of the ribs showed a fracture of the last rib on the left side, without displacement. X-rays of the cervical and dorsal spine showed complete obliteration of the intervertebral space between the fourth and fifth dorsal vertebrae. In the lateral view the body of the fourth dorsal vertebra showed an indefinite area of rarefaction in its central portion, and the posterior margin of it and of the fifth were ill-defined. Irregular areas of calcification were seen in the posterior central portions of both vertebral bodies. The appearance was thought to be consistent with a destructive process involving the fourth and fifth dorsal vertebrae, with calcification in its central portion. No soft-tissue shadow was seen. There was no evidence of disease in the skull. Lipiodol injected

into the cistern remained in the skull until the fourth day, when about a quarter of it was found in the terminal sac. An attempt to bring it into the cervical canal showed a definite temporary stop at the upper border of the fourth dorsal vertebra. There was no definite cap formation, and from that point the lipiodol trickled through a slightly irregular canal along the left side of the spinal canal at the level of the third vertebra and on into a normal cervical canal.

Combined cistern and lumbar punctures showed delayed rise and fall of the lumbar pressure on jugular compression. The compression finally elevated the lumbar pressure to only 240 mm. of water, whereas the cistern pressure rose to 350. The spinal fluid from both locations contained normal numbers of cells and normal amounts of sugar and protein.

An operation was performed on the twentieth day.

Differential Diagnosis

Dr. Tracy J. Putnam*: Of course we have an advantage in approaching this history as we always do over the man who saw the patient on the ward, because we know that she was operated on and that she went through Dr. Mallory's hands. These two facts give us a broad lead that her signs and symptoms are not hysterical manifestations.

"During the ten months before entry this feeling of numbness gradually extended up her legs and trunk until it reached the level of her breasts." Some people would say this is an example of an ascending neuritis. As a matter of fact, I think that is not justified on the history. We find that in many instances sensory and motor symptoms ascend, even when they are caused by a single lesion, so that this is not evidence against a transverse lesion which is suggested by some of the symptoms which we shall encounter later on.

"She also had some hypersensitivity and pruritus in the area of numbness. . . ." We do not learn whether this was in her feet or simply at the upper edge of the numb area, which is a commoner location.

"On two occasions she fell to the floor after suddenly standing up." This, in general, could perfectly well go with a lesion of sensation.

It was kind to put in a note about cataract because we should be tempted to connect the trouble in the eye with that in the spine, but presumably they were of different origin.

"About four months before entry she began to drag her right foot, and some time after that she began to have involuntary twitching movements of her legs." The description does not tell whether there were fibrillations in her leg — presumably not. The twitchings were probably the involuntary contractions of greater or lesser magnitude that are common with transverse lesions of the cord.

It is stated that a painful lump appeared on the left chest. Apparently this did not persist, and we find nothing to tell us what the nature of it was. This would be extremely difficult to explain on a neurologic basis, but if we glance ahead we see that x-rays showed fracture of the rib on the left side without displacement. Nothing more is said about it and we have no evidence to suggest that this was a pathologic fracture. It is curious to have spontaneous fracture occur without a pathologic process in evidence. "The pain was of brief duration but soreness persisted in the local area, and she stated that a large swelling appeared there, which gradually subsided." The whole thing could quite well go with fracture.

It is quite important that there were no symptoms of tuberculosis and no changes in the lungs because, as we shall see, the x-ray picture suggests the possibility of a tuberculous process in the vertebrae.

"The fundus of the right eye showed pigmentation in the macular region, and just temporal to this were two, small, grayish cystic areas invaded by small blood vessels and surrounded by fresh hemorrhages." This is a very curious finding, indeed, and I am at a loss to explain it. It certainly is not the description of any neuritic process.

"The visual fields were normal." The degenerations must have been very small, since they did not interfere with vision.

"The gait was somewhat unsteady, and there were involuntary twitching movements of the legs, especially on the right." From this I think we can judge that they were not fibrillations but active contractions of large groups of muscles. The atrophy may go with the long weakness and decreased usage of the legs.

There were pyramidal-tract signs on both sides. All forms of sensation were diminished below the eighth dorsal vertebra. In short, I think we are justified in saying she had a gradually progressive paraplegia, which might well be explained on the basis of a single lesion. The only questions might be the curious spots in her eyes and the fact that she sustained a pathologic fracture of the left twelfth rib. There is nothing in the chart of any great significance. I notice that Bence-Jones protein was looked for and not found. The red-cell count is only slightly low, and I believe that this could be taken as evidence against a malignant

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tumor, which is perhaps suggested by the finding which we shall read presently. It seems to me that the other laboratory findings are not of particular importance in this case except that the cystometrogram showed a hypotonic curve. I am at a loss to understand this because the other signs we have had so far have pointed to a lesion high up in the dorsal region, and yet there is a relaxed flaccid bladder where we should expect, if anything, a spastic one.

Then we come to the x-rays. From the neurological examination we should expect a lesion somewhere in the region of the transverse process of the eighth dorsal vertebra. It is very difficult to tell from the history whether this is an intrinsic degenerated lesion or an extrinsic mass compressing the cord. I may say that in cases of this character pruritus and hypesthesia at the level of the lesion are sometimes taken to suggest the presence of an expanding lesion, but we have had a number of cases in which they have been present with an intrinsic myelitis of the cord. In the physical examination no record is made of tenderness or limitation of motion, and it is astonishing if this did not occur.

The coincidence of the x-ray changes with the findings pointing to the cord certainly suggests that the two are related. One expects to find the lesion one, two or even three vertebrae above the involved segment in the spinal cord. No soft-tissue shadow was seen. Certainly the description and the plates themselves are not very suggestive of a tuberculous lesion, although the description might lead one to think of it. There is not enough kyphosis. Apparently this is not so dense a compression as to prevent lipiodol's passing through. Of course it is not at all unusual — in fact, it is quite customary — for a combined puncture to show a block which lipiodol does not demonstrate. The combined puncture is a far more delicate means of showing compression and narrowing of the spinal canal. The fluid showed a normal number of cells and normal sugar and protein. This is merely of negative importance. It is interesting that the protein was not increased.

Taking the history as a whole, it seems to me that we doubtless have a compression of the cord in the region of this abnormality of the vertebrae, and the whole diagnosis hinges on the nature of the process which we see in the x-ray picture. I do not know that tuberculosis can be definitely ruled out, but it certainly seems unlikely in view of the appearance of the films. Another possibility, of course, is a metastatic process. The appearance of the lesion to my eye, however, is not at all typical of metastatic cancer. There are no recognizable punched-out holes with proliferation of the bone around them, such as we are accustomed to find; but the appearance of metastatic processes does not always run true to form. The fact that she had an enlarged liver and pigmented nodules in the eye grounds might be taken as a suggestion in that direction, but on the whole I am against such a diagnosis and much more in favor of thinking that the difficulty is due to a local neoplasm.

Can we go farther and predict the type? It surely is not the ordinary fibroma or glioma arising in the cord. Neither of them ever damages the bodies of the vertebrae. Of the other tumors I should most seriously think of hemangioma. The x-ray picture of hemangioma is that of destruction with calcification which sometimes can be traced as being in the wall of the blood vessel; I suppose we could conceive that this spot in the film represents a blood vessel. On the other hand the picture is not wholly typical. Another possibility is some such tumor as reticulum-cell sarcoma, but this rarely causes destruction of the vertebrae. On the whole, I am in favor of a diagnosis of hemangioma of the vertebral body with compression of the cord, and I should be inclined to skip or side-step the question of the spots in the eye grounds. I have no particular explanation for the apparently spontaneous fracture of the rib.

X-Ray Interpretation

Dr. George W. Holmes: I am a little more in favor of tuberculosis, so far as the x-ray alone is concerned, than is Dr. Putnam. I think this lesion is rather consistent with tuberculosis. The intervertebral disk is destroyed; it is rather unusual for any neoplasm of the spine to destroy the disk, and certainly unusual for an angioma to do that without quite extensive destruction of the body itself. Then we have a shadow of increased density which might be something in the tumor — a calcium deposit which so frequently accompanies tuberculosis. If she had — and I presume from these films she did have — a definite block, that is somewhat difficult to explain on the basis of tuberculosis without kyphosis.

Dr. Putnam: Might I add a word on the basis of Dr. Holmes's remarks?

His opinion alters the situation a good deal. Of course a negative history and negative findings do not rule out Pott's disease. One could get a compression of the spinal cord from Pott's disease without kyphosis simply from the sac of fluid pressing on the spine. It is rather unusual in this community to operate on Pott's disease, at least without a long period of rest, and that, I may say, rather influenced my judgment.

As to the spinal-fluid findings, in the presence of neoplasm one may or may not find increased

protein, and certainly an increase of cells is often found.

Dr. James B. Ayer: There was another x-ray picture, which showed a questionable, healed tuberculosis in the lungs, that does not seem to be in this collection. It influenced us at the time. When we decided to operate, it seemed as if we might be dealing with a primary lesion of the second vertebra. In spite of the fact that tuberculosis was the most probable diagnosis, the patient was advancing in her paralysis and her sensory disturbance, and it seemed that we could not wait, even if we had wanted to, to give her the advantage of prolonged rest. When we operated with rather a forlorn hope, we all believed that the lesion was something amenable to surgery and that it was a vertebral disorder which had not been diagnosable by x-ray.

Dr. James C. White: We could not carry our preoperative diagnosis any farther than Dr. Putnam has. I thought it was tuberculosis and talked it over with the orthopedic service. They definitely believed that the x-rays were not typical of tuberculosis, so we made a preoperative diagnosis of compression at the fourth thoracic vertebra and did a laminectomy of the third, fourth and fifth vertebrae. You could see that the dura was pulsating normally, and on opening the dura it first appeared as if it were going to be a negative exploration. Then looking a good deal closer along the third thoracic posterior root I found that this root was pushed up posteriorly. On removing the arachnoid very carefully, there was a little yellowish tumor, which was quite avascular. In order to get a good exposure I had to take off part of the second lamina as well. On dissecting around it, it was seen that the tumor was pressing against the spinal cord on the right side. The dural attachment covered a base of 2 cm. and was so firm that I had to separate the tumor from the dura by sharp dissection. Then it was apparent that it was going around the dentate ligament and compressing the cord anteriorly as well. The tumor measured 2 cm. in length and 7 or 8 mm. in breadth. Finally, I fulgurated its attachment and felt very carefully to find out if there was any lesion that was growing extradurally and involving the bone: there was nothing to suggest it.

Preoperative Diagnosis

Compression of spinal cord at level of the third and fourth dorsal vertebrae.

Dr. Putnam's Diagnoses

Compression of spinal cord from extradural neoplasm.

Hemangioma of vertebra.

Anatomical Diagnosis

Meningioma of spinal dura.

Pathological Discussion

Dr. Tracy B. Mallory: Dr. Kubik was called to the operating room and made a frozen section diagnosis of meningioma. The tumor was well defined and full of psammoma bodies. Neither Dr. Kubik nor I could remember an instance of spinal meningioma that invaded the vertebrae. On the other hand we frequently see cranial meningiomas that invade the skull,—both the cranium and base of the skull,—and I can see no reason why an occasional one in the spine should not invade bone extensively. We have no actual proof that the meningioma invaded the vertebrae, but it seems to me that that is a strong probability.

Dr. White: She had a smooth convalescence and had little trouble with her bladder. I do not know why she had a hypotonic bladder before operation. She should have had a hypertonic one, as Dr. Putnam suggested. She recovered motor power in her legs.

Dr. Ayer: How has she been since discharge?

Dr. White: She is walking with a normal gait, and sensation in her legs is markedly improved.

CASE 24352

Presentation of Case

A fourteen-year-old, Jewish schoolgirl entered the hospital complaining of pain in the left flank and cough.

She was perfectly well until five months before entry, when she had a sudden attack of pain in the right flank. The pain was not severe enough to double her up. It was steady and persisted unchanged until two months before entry, when it entirely disappeared. The pain was aggravated by motion. About two weeks after the onset of the pain she developed a dry, non-productive cough, which lasted two weeks. The coughing aggravated the chest pain. She had a fever with a temperature which often rose as high as 102°F., but no dyspnea, hemoptysis or other cardiorespiratory symptoms. A diagnosis of pleurisy with effusion was made, and two thoracenteses done about a month after the onset of the illness yielded a total of about 150 cc. of bloody fluid. Tuberculin tests at that time were negative. She spent most of the time in bed. Several x-ray examinations showed persistence of the fluid in the left chest. Her strength remained good, and during the five months before entry she gained 25 lb. She had no genitourinary or gastrointestinal symptoms. Her

past history and family history were noncontributory. She had not been exposed to tuberculosis.

Physical examination revealed an obese, well-developed girl in no obvious discomfort. In the lower two thirds of the left chest there were dullness to flatness, distant breath and voice sounds, absent fremitus and no rales. The heart was perhaps slightly displaced to the right, and a rough systolic murmur could be heard at the left base below the ribs. The sounds were normal, and there were no thrills. The blood pressure was 104 systolic, 75 diastolic. The abdomen was negative, and no superficial lymph nodes could be palpated.

The temperature was 98.6°F., the pulse 95. The respirations were 20.

The urine examination was negative. The blood showed a red-cell count of 5,050,000 with 70 per cent hemoglobin and a white-cell count of 11,900 with 56 per cent polymorphonuclears, 34 per cent lymphocytes and 9 per cent monocytes. A 1:10.000 tuberculin test was negative.

During the first three days, four chest taps were done, each yielding grossly bloody fluid which clotted rapidly. Cultures of the fluid were sterile, and no tumor cells could be identified. Guinea-pig inoculation for tuberculosis was negative.

On the sixth day an x-ray of the chest showed a huge tumor mass occupying the region between the left sixth rib and the transverse process of the second lumbar vertebra. It displaced the heart, mediastinum, spleen, kidney and stomach, and its upper border was covered by fluid in the chest. The left tenth rib was situated in the center of the tumor mass, and its lateral half was eroded, irregular and distorted. The ribs above and below it were widely separated. There was no calcification in the soft tissues, and no evidence of metastases in the upper part of the left lung, in the right lung or in the visualized bones. An intravenous pyelogram showed displacement of the left kidney, without deformity. The outline of the tumor mass was sharp, and it lay distinctly above the kidney but did not involve it. The left transverse process of the first lumbar vertebra was indistinct and was thought either to be eroded or to show an anatomic variation. The right kidney, ureters and urinary bladder were not remarkable.

On the eighth day an operation was performed.

Differential Diagnosis

Dr. Joseph Garland: The history is slightly confusing to me. The patient apparently entered the hospital complaining of pain in the left flank and cough, while in the next paragraph we find that the attack she had five months before entry was in the right flank, and thereafter neither flank is mentioned. A little later, we are told that she had cough, which aggravated the chest pain. That is the first and only mention of pain in the chest. So, apparently, both of these symptoms, the pain and the cough, which are the only symptoms complained of, had ceased two months before entry to this hospital. During part of that time she had a fever, and she had sufficient chest signs, even though the symptoms did not persist, to warrant chest taps, at each of which bloody fluid was obtained. Tuberculin tests were negative, and later, during her hospital stay, we find that the tuberculin test was again negative; eventually guinea-pig inoculation was also negative. In the hospital four more chest taps were done within a comparatively short period, — three days, — each of which again yielded bloody fluid which clotted. The question comes up as to whether this bloody fluid was traumatic in origin or whether it actually was the fluid present in the pleural cavity. I should be inclined to feel that it was unlikely that five or six taps should be done each yielding blood of traumatic origin, and I consequently believe that the fluid in the chest cavity was bloody.

After six days in the hospital we have a rather revealing chest x-ray, although what it reveals I do not know: "A huge tumor mass occupying the region between the left sixth rib and transverse process of the second lumbar vertebra."

X-Ray Interpretation

Dr. Aubrey O. Hampton: The first films were taken three months before the patient entered the hospital, during the time she was having chest taps for pleural fluid, and she entered the hospital with about the same picture that she had had three months previously. On the original films you can see an abnormal tenth rib on the left side, which at the later examinations is grossly abnormal and shows moth-eaten bone destruction and new-bone formation. This rib is considerably separated from the adjacent ribs. In order to determine the extent of this density on the left we laid the patient on her back and the fluid flowed away from what appears to be a very high left diaphragm. The diaphragm or mass is at the sixth rib on the left side. The fundus of the stomach is displaced downward and medially from the tenth rib to the first lumbar vertebra a distance of 15 cm. from its normal position. The left kidney is displaced downward and rotated, and here we see the inferior margin of the mass. It is smooth and sharp, and there is no evidence that it arises from the kidney, but there is good evidence that it arises from the rib.

Differential Diagnosis (continued)

Dr. Garland: The question still is, Is this an intrathoracic mass or an abdominal mass? The printed interpretation made me feel that it was above the diaphragm until Dr. Hampton raised the possibility of a very high diaphragm on the left, which would place the whole process below it and would help to confuse the picture.

There are various possibilities we have to consider. Some of these ought to be eliminated fairly readily. The general character of the process and the fact that we have had two negative tuberculin tests and a negative guinea-pig test practically rule out tuberculosis. I think that the blood in the chest fluid is part of the picture and not blood that is due to the needle. That brings us down to the consideration of various tumors.

What types of tumor are most likely in a fourteen-year-old child? There are none particularly likely. Primary tumors of the pleura or of the lung are both very rare. In either case they are almost sure to be some type of sarcoma. The tumors originating in the mediastinum are rare; the more likely ones include lymphoma, thymus tumors and, occasionally, dermoid cyst or teratoma. If we were to assume, because of the x-ray, that the tumor arose in the rib, we should classify it as a sarcoma, and such is my diagnosis. It shows no metastases, but I believe a round-cell sarcoma may grow to considerable size without metastases.

Dr. Tracy B. Mallory: You place the primary site in the rib?

Dr. Garland: Yes.

Dr. Channing C. Simmons: I believe that the group of malignant lymphomas should be considered and that some of the rarer tumors of the kidney would fit the case. Tumor of the rib would not produce so much marked pleural involvement and would not give so much fluid in the chest as she had.

Dr. Mallory: Is it possible for an osteogenic sarcoma or a Ewing's tumor to be primary in a rib?

Dr. Simmons: Yes. A Ewing's tumor usually gives a slight rise in temperature and may cause a slight rise in white count. Moreover, by x-ray one expects true bone destruction and practically no new-bone formation.

Dr. William B. Breed: We know that if this patient was operated on they must have made up their minds before operation whether it was below or above the diaphragm, and I suppose Dr. Hampton must have been consulted in that decision. The opinion of the x-ray man must have been valuable in this case.

Dr. Hampton: I think if you looked at the film closer, Dr. Simmons, you would be fully convinced the lesion is in the rib. The destroyed rib is in the center of the mass, so we believe it was primary in the rib—a primary bone tumor. I have seen a somewhat similar picture caused by tuberculosis of the rib, accompanied by a large soft-tissue mass, a cold abscess; that is the only other possibility.

Preoperative Diagnosis

Tumor (unclassified), left chest.

Dr. Garland's Diagnosis

Tumor, probably sarcoma, probably osteogenic.

Anatomical Diagnosis

Primary malignant tumor of bone, unidentified.

Pathological Discussion

Dr. Mallory: As a matter of fact, I am the person on the spot in this case, because I cannot tell you what the tumor was. This patient's picture was very interesting, however. She was seen by Dr. Churchill who thought the tumor was probably primary in the ribs, but inoperable. She was then turned over to Dr. Hampton for x-ray treatment, which he gave, the tumor shrinking incredibly. A few weeks afterward Dr. Churchill decided that the tumor was operable, so he excised it, taking out three ribs. We found that the tumor had destroyed the middle one of the three ribs and pressed against the other two but had not involved them, so I think there is no doubt but that it was primary in the tenth rib. We had a biopsy before the original x-ray treatment and were unable to make a diagnosis. When the tumor was resected we made at least twenty sections from different areas and could not find a viable tumor cell anywhere in it—nothing but necrotic material with inflammatory reaction at the periphery, granulation tissue and old blood. Perhaps Dr. Hampton will proceed with the story.

Dr. Hampton: We have been following the patient for four years and she has remained clinically well. This is the film showing the disappearance of the mass within three months under x-ray treatment—complete disappearance of the mass both in the chest and abdomen after 5700 r of deep irradiation, which is a considerable dose, given over a long period of time. These are postoperative films taken in 1937. Up until then she showed absolutely nothing clinically or otherwise but at that time a 1-cm. nodule developed in the outer aspect of the right lung and at present she has another posteriorly. She thus has recurrent disease in the opposite lung.

DR. MALLORY: This tumor has a very unusual appearance. It is not, like most unclassifiable tumors, totally undifferentiated and highly malignant-looking but is something that one feels one should be able to recognize. It consists of anastomosing cords of tumor cells which twist about very dilated sinusoids — spaces with a capillary lining but of much greater than capillary proportions. The general architecture is very much like that of an endocrine gland. We have seen one other tumor that seemed to be identical in appearance which was also primary in bone, in that instance in the jaw. It was treated rather heavily with radiation but did not respond. I have shown the sections to a large number of pathologists, and no one is willing to commit himself as to the diagnosis. The suggestion is that it is a type of tumor that is apt to be primary in bone and does not occur elsewhere. I do not know what it is.

DR. SIMMONS: Does it suggest a reticulum-cell sarcoma in any way?

DR. MALLORY: No; the architecture of the tumor is entirely different, and the cells do not look like histiocytes.

DR. JOHN H. TALBOTT: How much involvement was there of the diaphragm and adrenal?

DR. MALLORY: It was necessary to remove a part of the diaphragm in order to remove the rib. The disease was at the extremity of the tenth rib but the diaphragm, so far as we could make out, was not involved. The adrenal was not examined.

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ARE CHIROPODY AND OPTOMETRY UNETHICAL PRACTICES?

In a recent issue of the *Journal of the American Medical Association* (*111*:49, 1938), there is the report of the presentation to the House of Delegates of the American Medical Association of a resolution on teaching in schools of chiropody. The resolution declares that teaching in such schools by members of the American Medical Association is unethical. The matter was referred to the Judicial Council for its consideration.

The grounds for this view are twofold. It is first stated that the resolution is supported by an analogous decision by the American Medical Association in regard to teaching in schools of optometry. It is then brought out that teaching in cultist schools and addressing cultist societies have been condemned by the Judicial Council of the American Medical Association for its members. One must conclude that both optometry and chiropody are regarded as cults by the American Medical Association, but such a classification does not seem to be justified.

What is the nature of a cult in medicine and why should the pursuit of a cult be regarded as unethical? The taint of the cult from the point of view of science is that its pursuit is associated with an extravagant emotional reaction, so extreme that it passes the bounds of reason or common sense. It has the implication of worshiping some element not deserving of worship. It is the unbalance of the cultist that is scientifically objectionable, and it is the antagonism of science to obscurantism that finds its expression in the dogma that it is unethical for the physician to assist in any way in the promulgation of a cult in medicine.

But are optometry and chiropody cults? Either of these branches of the healing art may be practiced by physicians. There is nothing esoteric about chiropody. If a qualified physician chooses to limit his practice to the field beyond which the chiropodist must not go, he may properly do so. It may be that chiropodists have made little contribution to the science underlying the art which they practice. It may be that some chiropodists are unethical; but so are some physicians who have nothing to do with chiropody. It may be that there is vast need for improvement in chiropody. Medicine also offers opportunity for improvement. Neither the whole of medicine nor any part of it is intrinsically unethical. "Unethical," as applied to the practice of medicine, refers to the way in which it is practiced.

There apparently exists within the American Medical Association a misconception of the definition of a cult and of the nature of chiropody and optometry. Action based on a misconception of the facts is always fraught with danger. It was a misconception and misunderstanding, by physicians, of the nature of dentistry which led to the development of this separate profession — a separation because of which both medicine and dentistry are still suffering severely.

Chiropody is a branch of medicine, the importance of which is becoming more widely re-

alized. It has become an important adjunct in the diabetic clinic. One may go even farther and say that proper care of the feet has become an integral part of the adequate treatment of the diabetic patient. The development of schools of chiropody in complete independence of schools of medicine is to be deplored, as harm will result to both branches of the healing art. Instead of trying to separate the two more widely, every effort should be made to bring them closer together, and the participation of qualified physicians in chiropodic education should be regarded not only as *ethical* but as *necessary* for the proper development of the art and of the underlying sciences. It is to be hoped that the Judicial Council of the American Medical Association will not recommend the adoption of this resolution, and that the former action condemning teaching in schools of optometry will eventually be revoked.

PROGRESS IN DIABETES CONTROL

Diabetes, according to a recent issue of the *Statistical Bulletin* of the Metropolitan Life Insurance Company, continues to be an outstanding example of the progress which modern medical science has made in combating disease. It shows also how international in scope these scientific methods may be. The pancreatic origin of diabetes was first suspected by a Frenchman in 1846; it was proved by two Germans in 1889. In 1901 an American and in 1902 a Russian showed independently the involvement of the islands of Langerhans. Canadians discovered insulin in 1921, and in 1935 a Dane demonstrated the value of adding protamine to insulin.

The study of diabetes has always been progressing; two landmarks in this progress, however, have been the discoveries first of insulin and then of protamine-zinc insulin. The most immediate and lasting effect of the use of insulin on the diabetic situation has been the drop in mortality among children and young people. With this there has been, obviously, an increase in the rate among older individuals, indicating the increased frequency of discovery of cases, hitherto overlooked, — particularly of mild cases in later life, — and the constantly growing number of persons at older ages, among whom the frequency of onset of diabetes is the highest. With an actual unquestioned increase in the disease, its hardships have been ameliorated, and its dangers reduced.

Protamine has introduced a new era in the treatment of the disease. More diabetics are willing to take insulin, for it now needs to be injected only once a day. Diets may be still further liberalized. Enlargement of the liver is tending to disappear. Coma is less frequent, and surgery has been made safer. The onset and progression of arteriosclerotic complications seem to be delayed. These are some of the benefits that protamine has brought in little over two years of use.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

Raymond S. Titus, M.D., *Secretary*
330 Dartmouth Street
Boston

Ruptured Uteri

For nearly two years the Section of Obstetrics and Gynecology of the Massachusetts Medical Society has presented weekly in the *Journal* actual cases illustrating antepartum hemorrhage. Differential diagnosis and proper and improper treatment have been emphasized. Beginning in the following issue a series of cases on ruptured uteri will appear. These will cover spontaneous and traumatic rupture of the uterus.

Spontaneous rupture of the uterus may occur before or during labor. The former is of course much less frequently seen than the latter, but up to 1923 over 100 cases had been reported. Interstitial pregnancies, rupture of cesarean-section scars and bi-cornate uteri are by far the commonest causes of rupture during pregnancy. It has been suggested that rupture can be attributed to a hardened degeneration of the uterine muscle, to invasion of the uterine muscle by fetal elements or to extensive round-cell infiltration. Most of these

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

cases occur in multiparae. Trauma and infection following curettage and the removal of a previously adherent placenta are suggested as causes of this hardened degeneration. Spontaneous rupture of the uterus during pregnancy always occurs in the upper segment of the uterus. The patient presents the picture of an acute abdomen, plus that of shock and hemorrhage. Hemorrhage of course need not be the salient point if the amount of blood is so slight that the exact cause is not determined until the abdomen is opened and the rupture found. The important point to appreciate is that an immediate operation is necessary. In a differential diagnosis one must consider twisted ovarian cyst, acute appendicitis and acute intestinal obstruction. If the patient has had a previous cesarean section that fact should suggest the correct diagnosis. The treatment, depending entirely upon the extent of the rupture, varies from suturing the separated uterine muscle to supravaginal hysterectomy. One must bear in mind that ruptures do occur in cases in which low-cervical sections have previously been performed. The incidence of rupture in cesarean scars has been estimated as high as 3 per cent. This is probably too high; but no matter how smooth a convalescence, no matter who the operator, the possibility exists.

Spontaneous rupture of the uterus during labor but before delivery is uncommon, but a true emergency it is. Lobenstine[1] in 1909 reported 28 spontaneous ruptures in 60,000 cases, and Ossendorff[2] in 1912 reported 10 in 12,000 deliveries. As in rupture during pregnancy, the tear is in the body of the uterus and occurs much more frequently in multiparae than in primiparae. It is frequently caused by an abnormal Bandl's ring. Its diagnosis is made certain by the history of a patient who, after a severe pain, faints and ceases to have contractions; the presenting part may no longer be in the pelvis, the fetal heart sounds are absent, and the patient presents a picture of profound shock. The symptoms of spontaneous rupture may not occur until the time of delivery, and in such a case the rupture is usually in the lower uterine segment and starts from the giving way of a scar in the cervix from a previous delivery. The diagnosis is suggested by the patient's condition of collapse and hemorrhage and is confirmed by putting one's hand into the uterus and feeling the rent in the lower segment. In either case, laparotomy is indicated as soon as the diagnosis is made. Whether a hysterectomy is to be performed or the uterus sutured depends upon the conditions that are found.

Operative deliveries undoubtedly cause the greatest number of ruptured uteri. Version done in a dry uterus, — a very serious performance, — delivering a patient whose cervix is not well dilated, and the delivery of a patient with a contraction ring, all are predisposing causes to this condition. Attempting to push a hydrocephalic head through the lower segment is often followed by a low rupture. After any serious operative delivery the cervix should be inspected and, if torn, should be ligated; the fundus should be explored, and if a rent is discovered, laparotomy should follow.

In the succeeding numbers of the *Journal,* cases covering this condition will be presented. Its diagnosis, its differential diagnosis and its treatment — operative and medical — will be emphasized.

REFERENCES

1. Lobenstine, R. W.: Rupture of the uterus during labor: a study based upon the cases treated in the three divisions of the New York Lying-In Hospital. Am. J. Obst. 60:819-852, 1909.
2. Ossendorff, K.: *Ueber Uterusruptur und ihre Behandlung.* 60 pp. München: Müller & Steinicke, 1912.

DEATHS

AUSTIN — Arthur E. Austin, M.D., 110 Marlboro Street, Boston, died at his summer home in Windham, New Hampshire, August 22. He was in his seventy-eighth year.

Born in Boston he graduated from Bowdoin College in 1883 and received his degree from the Harvard Medical School in 1887. Later he received a degree from the University of Berlin. Before returning for further work at Harvard in 1892-1893 he studied at Heidelberg University. He was professor emeritus of the theory and practice of medicine at Tufts College Medical School.

A member of the Tufts faculty when the school was founded in 1893, he taught there until 1906, returning in 1910. He taught two years at the University of Texas and was elected adjunct professor of physiological chemistry at the University of Virginia in 1908. During the World War he was commissioned a major in the army medical corps.

He was the author of *Manual of Clinical Chemistry* and *Diseases of the Digestive Tract and Their Treatment.* Dr. Austin was a member of the American College of Physicians and a former fellow of the Massachusetts Medical Society.

His widow and a son survive him.

BARRON — Maurice E. Barron, M.D., of West Newton, died August 22. He was in his forty-seventh year.

Dr. Barron received his degree from the Tufts College Medical School in 1914. He began practice the following year. He was professor of clinical surgery at Tufts College Medical School.

A fellow of the American Medical Association and the American College of Surgeons, he was also a member of the Massachusetts Medical Society.

His widow, two sons, and a daughter survive him.

MISCELLANY

LIST OF APPROVED RESPIRATORY PROTECTIVE DEVICES

A list of respiratory protective devices approved by the Bureau of Mines, United States Department of the Interior, has just been published.

To insure that safe, satisfactory and reliable respiratory protective devices will be available the Bureau of Mines

has established schedules of minimum requirements for oxygen breathing apparatus, gas masks, supplied-air respirators (hose masks, airline respirators and abrasive blasting helmets, hoods, or masks), and mechanical-filter respirators.

Manufacturers of such devices may submit their equipment to the bureau for approval under the requirements of these schedules. Not only must the manufacturer's devices meet the bureau's requirements, but he must have satisfactory equipment for controlling their quality. If all the requirements and tests of the schedules are met, the manufacturer is granted a certificate of approval which states that the equipment has passed the schedule of requirements and gives the limitations of the apparatus. He may advertise these devices as approved by the Bureau of Mines; the bureau may rescind the approval for cause.

Lists of approved devices are published periodically to make available to the public a ready source of information on approved respiratory protective equipment. The present list designated as Information Circular 7030, "List of Respiratory Protective Devices Approved by the Bureau of Mines," by H. H. Schrenk, contains all devices approved to May 20, 1938. Copies of this circular may be obtained from the Bureau of Mines, Washington, District of Columbia. — *U. S. Department of the Interior.*

NEW MEASURING ROD FOR COMMUNITY HEALTH SERVICES

The American Public Health Association has recently issued an *Appraisal Form for Local Health Work*. The 200-page document is the result of studies in measuring community needs and community health activities by committees of the association which have been continuously engaged in this work for eighteen years.

The form is primarily a measuring rod for community health services. Through the use of a carefully arranged survey schedule the principal health problems of the community are brought to light and defined. The program of community health service in the several fields is analyzed against this background of social and health conditions. The health services as performed are studied in relation to the need, with the result that the relative adequacy of public-health work is judged with some consideration of the magnitude and type of local problems.

Heretofore the association has issued separate schedules for urban and rural work. Advances in rural health practice in the last few years, however, have been so great that the association believes that issuing separate schedules for the measurement of urban and rural health service tends to accentuate the differences in practice which are rapidly disappearing.

The form is divided into three major sections: a survey schedule for studying a community's health facilities, a schedule for appraising the local health activities, and a list of significant health indices. The association urges the use of the form by health workers and communities to evaluate periodically and impersonally their public-health programs and to determine the fields in which change of emphasis may be needed for more effective work.

CORRESPONDENCE

NEW REPORTABLE DISEASES

To the Editor: Because of the occasional discovery of cases of psittacosis, Rocky Mountain spotted fever and tularemia and of the desirability of having these diseases officially recorded, the Department of Public Health, on August 9, declared these diseases dangerous to the public health.

This action makes it necessary for physicians to report them to their local boards of health, which in turn, relay the reports to this department.

ALTON S. POPE, M.D.,
Deputy Commissioner of Public Health.
State House,
Boston.

HOME TREATMENT OF TUBERCULOSIS

To the Editor: In the August 11 issue of the *Journal*, Dr. William S. Streker, of Providence, takes issue with Dr. Volk regarding the home treatment of pulmonary tuberculosis. Dr. Streker states that "we cannot by any stretch of imagination match in the home the benefits of treatment to be obtained in the modern sanatorium, with its trained personnel and complete facilities."

According to Dr. Streker, there is something magic about sanatoriums which cannot be duplicated in the home. Maybe the sanatoriums in Rhode Island are the acme of perfection. Here in Massachusetts we have some of the best sanatoriums in the country; we also have some that are far from what one might desire and some that do not measure up to any standard. Possibly Dr. Streker has never heard of politics.

There are still a few self-respecting individuals left who can afford to pay a private physician who is well trained in the treatment of pulmonary tuberculosis. Not all of them have remained in institutions. Given a patient who can afford it, all the modern treatment and laboratory tests can be carried out in the home by the experienced physician. At best, the average time a patient spends in a sanatorium is from six to eight months. Hence, home treatment must remain an integral and important part of therapy.

One certainly cannot treat a fool or a poor man at home, but with intelligent co-operation, ideal sanatorium treatment can be duplicated, in conjunction with general hospital care if surgery is required.

MOSES J. STONE, M.D.
330 Dartmouth Street,
Boston

NOTICES

TESTIMONIAL DINNER FOR DR. CHADWICK

A testimonial dinner for Dr. Henry D. Chadwick will be given by his friends in the Sheraton Room of the Copley-Plaza at 7:00 p. m., Thursday, September 8. Because of the proximity of the date, no personal invitations will be mailed; all of Dr. Chadwick's friends are cordially invited to attend. Those planning to be present are requested to obtain tickets from or to notify Miss Helen Condon, Massachusetts Department of Public Health, State House, Boston; the charge for the dinner has been set at three dollars per plate.

SPECIAL SESSION OF HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION

In compliance with the official request of members of the Board of Trustees that the House of Delegates be convened in special session, I, as speaker, under authority of

Chapter III, Section 2, of the by-laws hereby officially call the House of Delegates of the American Medical Association to convene in special session in the City of Chicago, State of Illinois, at 10:00 a. m., Daylight Saving Time, on September 16, 1938.

The business to be transacted at this special session shall be limited to the consideration of the national health program submitted to the National Health Conference recently held in Washington and to such other matters as may be submitted to the House of Delegates by the Board of Trustees.

The House shall remain in session, recessing from day to day, until its deliberations are concluded.

This call is issued August 26, 1938.

H. H. SHOULDERS, M.D., *Speaker*,
House of Delegates,
American Medical Association.

PSYCHIATRIC CONFERENCES, BOSTON CITY HOSPITAL

A psychiatric conference, led by Dr. Merrill Moore, will be given in the Out-Patient Department at the Boston City Hospital on the first Wednesday of each month during the next four months. The days will be September 7, October 5, November 2 and December 7. These conferences will begin at 11:30 in the morning and will be over at 12:30. They will take place in the Nerve Conference Room on the sixth floor of the Out-Patient Department and are open to any physician or interested person.

TUMOR CLINIC, BOSTON DISPENSARY

Each Tuesday and Friday morning from ten to twelve-thirty there is a meeting of the Tumor Clinic of the Boston Dispensary, a unit of the New England Medical Center. All kinds of tumors are seen, discussed, and when indicated, treated with radium and high-voltage x-ray.

Physicians are welcome to visit this clinic and bring patients for diagnosis.

AMERICAN PUBLIC HEALTH ASSOCIATION

The scientific program of the sixty-seventh annual meeting of the American Public Health Association in Kansas City, Missouri, October 25, 26, 27 and 28, which will engage the attention of more than 3500 of the nation's health authorities, indicates how closely the organization's Program Committee has been following national trends in public-health progress.

There is considerable emphasis on the five major diseases which are being attacked throughout the land with government funds. The grave problem of maternal and infant mortality receives its share of attention. A special session is devoted to a discussion of "Public-Health Aspects of Medical Care," which it is expected will be one of the most significant of the entire conference, with exponents of the National Health Program, spokesmen for organized medicine and representatives of the newly-recognized medical consumers — the public — on the platform.

The recruiting and training of public-health personnel for the specialized tasks which the expansion of health services is creating and for which trained workers are seriously lacking are major notes in the varied program. The health department as a business organization is a new concept which will be treated by the special-session method. Administrative procedures and accounting measures, including cost-accounting, will be considered. Many health departments are now on a parity with million-dollar enterprises, and special technics and formulas for the conduct of the big business of public health are definitely needed.

A feature of this great health congress is the Health Exhibit which enables ethical commercial organizations to familiarize health workers with products, apparatus, equipment and services which have utility in modern public-health practice and value in health protection and promotion.

For further information address: American Public Health Association, 50 West 50th Street, New York, New York.

PENTUCKET ASSOCIATION OF PHYSICIANS

The list of speakers and their subjects that have been scheduled for the 1938-1939 meetings of the Pentucket Association of Physicians is as follows: September 15, "What X-Ray Therapy Can Be Expected to Accomplish," Dr. Frederick W. O'Brien, Boston; October 13, "Autonomic Pharmacology," Dr. Abraham Myerson, Boston; November 10, "Allergic Eczema and Asthma in Children," Dr. Lewis W. Hill, Boston; December 8, "Cutaneous Eruptions Found Among Gainful Workers in Massachusetts," Dr. John G. Downing, Boston; January 12, "Progress in Medicine During the Past Two Years," Dr. Reginald Fitz, Boston; February 9, "Present Status of Fever Therapy," Dr. John G. Gibson, 2nd, Boston; March 9, "Some of the Outstanding Features of Hematuria," Dr. Harold A. Chamberlin, Boston; April 13, "Troubles as the Food Goes South," Dr. Chester M. Jones, Boston; May 11, "The Diagnosis and Treatment of Hemorrhage in the Last Trimester of Pregnancy," Dr. Frederick C. Irving, Boston; June 29, "Medicolegal Questions Based on an Agenda from the Association," Leland Powers, Boston.

The meetings except for that on June 29, which is open, will be held at the Hotel Bartlett (Tel. — Haverhill 3680), 95 Main Street, Haverhill, at 8:30 in the evening.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, SEPTEMBER 5

TUESDAY, SEPTEMBER 6

*10 a.m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

WEDNESDAY, SEPTEMBER 7

*11:30 a. m. - 12:30 p. m. Psychiatric Conference. Boston City Hospital. Nerve Conference Room, 6th floor, Out-Patient Department.

THURSDAY, SEPTEMBER 8

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

FRIDAY, SEPTEMBER 9

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

SATURDAY, SEPTEMBER 10

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Marshall N. Fulton.

*Open to the medical profession.

SEPTEMBER 7 — Psychiatric Conference. Boston City Hospital. Notice above.

SEPTEMBER 12-14 — American Association for the Study of Goiter. Page 545, issue of March 24, and page 145, issue of July 28.

SEPTEMBER 12-15 — American Congress of Physical Therapy. Page 946, issue of June 2.

SEPTEMBER 15, 1938-JUNE 29, 1939 — Pentucket Association of Physicians. Page 321.

SEPTEMBER 19-22 — Congress of the International Society of Surgery. Page 180, issue of August 4.

SEPTEMBER 19-23 — American Association of Railway Surgeons. Page 181, issue of August 4.

SEPTEMBER 26 and 27 — Symposium on Occupational Disease. Page 145, issue of July 28.

OCTOBER 3-15 — Inactive Status Training Course. Page 288, issue of August 25.

OCTOBER 7 and 8 — Conference on Rural Medicine. Page 180, issue of August 4.

OCTOBER 8 and NOVEMBER 15 — American Board of Ophthalmology. Page 282, issue of February 10.

OCTOBER 17, 1938 and FEBRUARY 20, 1939 — American Board of Internal Medicine, Inc. Page 216, issue of August 11.

OCTOBER 17-21 — Clinical Congress of the American College of Surgeons, New York City.

OCTOBER 24-26 — Academy of Physical Medicine. Scientific Session. Washington, D. C.

OCTOBER 24-NOVEMBER 4 — Graduate Fortnight of the New York Academy of Medicine. Page 181, issue of August 4.

OCTOBER 25-28 — American Public Health Association. Page 321.

NOVEMBER 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

MARCH 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

BOOK REVIEWS

Operative Gynecology. Harry S. Crossen and Robert J. Crossen. Fifth edition. 1076 pp. St. Louis: The C. V. Mosby Company, 1938. $12.50.

This work has been appearing in new editions, except the last, every five years since 1915, and to those who are familiar with it, no introduction or further recommendation is needed than the statement that each edition shows definite improvement over its predecessor. This is especially true of the latest. To those unfamiliar with the book it may be said that this is the best operative gynecology in English.

The typography is good, the index comprehensive, — though it could be made more useful by some cross references, — and the text well written, and there is a wealth of illustrations. In the discussion of so extensive a field, where an effort is made to present the best contributions by many operators, it is natural that differences of opinion exist. But in the matter of illustrations, there is only one object, to make clear what is represented. While very great progress has been made since the first edition in this respect, and some of the pictures are delightfully explanatory, there are still too many that even the legend does not fully clarify. Diagrammatic or semidiagrammatic sketches are better for teaching purposes than inadequate photographs. The resources of modern photography have not been utilized so fully as one would like to see in a work of this character.

Attention is properly called to the change in the treatment of "pelvic inflammatory disease" in the past quarter century. A costly lesson has been learned, but it might have been learned much earlier if more attention had been paid to the natural history of the disease. What will happen if one does not operate immediately in acute gonorrheal salpingitis or septic abortion or, to go outside the field of acute infection, in tubal pregnancy? The present work would be more helpful if this point of view were more frequently emphasized, and many lives would be saved.

There is a thorough consideration of radiotherapy, and under the unassuming title of "special disturbances," there is a discussion of some of the late effects. The reviewer believes that it is only fair to say that even today we know little of the late effects and that each year there is accumulating more evidence to impress our ignorance upon us. We need renewed and intensified efforts to secure the benefits and to avoid the harm of radioactivity.

Occasionally there have been introduced historical sketches which might perhaps with advantage be extended to additional topics. But references to the literature would be far more helpful and take little more space if they were made more specific as to date of issue of the periodicals.

The wealth of material is impressive and at points where differences of opinion are known to exist, the authors exercise, in general, sound judgment and give wise advice.

Alfred Owre: Dentistry's militant educator. Netta W. Wilson. 331 pp. Minneapolis: The University of Minnesota Press, 1937. $4.00.

The name of the late Alfred Owre has been known, probably, to comparatively few. Yet without question he was the leading dental educator of his day, the builder of one important dental school and the reorganizer of another — a man some of whose ideas on dental education may still require to be reckoned with. A militant idealist of somewhat radical views, inclined to be contemptuous of opposition, Owre could not but make some enemies. On the other hand, his obvious sincerity, his devotion to the causes he espoused and his broad cultural interests brought him many and staunch friends. He was a lover of art and of literature and came to possess one of the choicest and most valuable collections of cloisonné in the country. For recreation he preferred walking, and in the course of his life covered thousands of miles in various parts of the globe. Miss Wilson not only has managed to present the facts of Owre's picturesque life but also has succeeded admirably in conveying his personality. She is to be congratulated on an excellent work which anybody in the medical or dental profession should thoroughly enjoy.

Life and Letters of Fielding H. Garrison. Solomon R. Kagan. 287 pp. Boston: The Medico-Historical Press, 1938. $3.00.

Garrison, who died in 1935, was the outstanding medical historian and bibliographer of this country. Nurtured by Billings in the Army Medical Library, he tendered exceptional service to the *Index Catalogue* and the *Index Medicus,* as well as writing the most fundamental text in English on medical history, *An Introduction to the History of Medicine.* That a biography, or a number of biographies, of this man should be written would not be surprising. Although Garrison's work was extensively noticed in the *Bulletin of the Institute of the History of Medicine* for April, 1937, Dr. Kagan is the first in the field with a book-length biography.

Garrison's life is briefly reviewed, but the bulk of the book and its most important part consist of letters from Garrison to his various friends. His letters to Dr. E. C. Streeter, seventy-five or more in number, most reveal the man for they are intimate, informative and in every way typical of him. At times sharp, they illustrate Garrison's great gift of characterization of men and reflect his wide knowledge of events. For the preservation of these, the book is worth while. Some might have waited for a later date for publication, for many persons still living appear in the contents.

The book, privately printed, is well gotten up. There is a good index. Unfortunately, a good many minor errors have crept in.

There is a taste of Garrison here, but one wants much more. Without the Streeter letters, the book would indeed be a skeleton. With them a little flesh and blood has been added. One must wait, however, for a full life — a life which will be worthy of a great man.